The Journal of
NERVOUS and MENTAL DISEASE

VOL. 129, NO. 1
July, 1959
SERIAL NO. 920

SOME FACTORS ASSOCIATED WITH CONCORDANCE AND DISCORDANCE WITH RESPECT TO SCHIZOPHRENIA IN MONOZYGOTIC TWINS

DAVID ROSENTHAL, PH.D.[1]

One of the interesting issues raised by geneticists regarding schizophrenia is referred to as "the biological unity of schizophrenia" and "the specificity of the schizophrenic genotype." Planansky (14) has provided a brief review of the literature concerned with this problem. In the main, relevant research has involved attempts to establish any one or more of four points: 1) differentiation of the manic-depressive from the schizophrenic genotype; 2) identification of a non-hereditary subgroup within the domain of schizophrenia; 3) genetic differentiation of the main Kraepelinian subtypes; 4) genetic differentiation of typical, or "process," from atypical or "reactive" schizophrenia. Following Planansky, we may summarize this literature briefly as follows.

1. *Specificity of the manic-depressive and schizophrenic genotypes.*
   a. Rüdin (17) noted that manic-depressive psychosis rarely occurred in the children of schizophrenic subjects, but occurred rather often among these subjects' parents. Since he adhered to a strict specificity theory, he tried to explain this puzzling finding by postulating a complex Mendelian segregation and interrelation between the two illnesses.
   b. Schulz (18) found large numbers of schizophrenics among the children of two parents with affective psychosis, whereas among the children of two schizophrenic parents, manic-depressive psychoses were very rare. Both psychoses were thought to occur more frequently in the same families than would be expected by chance alone, and he thought that there might be a genetic relationship between the two psychoses.
   c. Penrose (12) used extensive data from the Ontario mental hospitals and found that the same family often included both types of illness.
   d. Kallmann (7) believed that confusion resulted from faulty diagnosis. He stated that when diagnostic criteria were sharply defined as in his study, there was not one dizygotic twin pair with a schizophrenic psychosis in one member and a manic-depressive psychosis in the other, nor a single manic-depressive index family with an authentic case of schizophrenia among the parents and siblings of the index cases. Unfortunately, he does not present figures regarding the reliability of diagnostic differentiation when his criteria are used.
   e. Planansky (14) pointed out that there has not been one clear-cut case of monozygotic twins where one twin was schizophrenic and the other manic-depressive. This statement must be taken with reservation, since affective psychosis in one twin is not rare when the other twin is schizophrenic. Problems of diag-

[1] Laboratory of Psychology, National Institute of Mental Health, Bethesda, Maryland.

nostic reliability are even more clearly at issue in such matters.

2. *Identification of a non-hereditary subgroup.*
   a. Such a subgroup was thought to be found by Bleuler (3). He thought of it as a separate disease within the group of schizophrenias. Planansky however thought that, statistically, the distinction did not appear to be substantiated in Bleuler's sample.
   b. Rosanoff, Handy, Plesset and Brush (16) also thought that they had evidence that hereditary factors were not necessarily involved in schizophrenia, since they believed that the frequency of schizophrenia was higher among their dizygotic cotwins than among siblings of schizophrenic index cases. Kallmann (7) found no such difference.
3. *Genetic differentiation of the Kraepelinian subtypes.*
   a. Kallmann (6) found a higher rate of schizophrenia among the children of hebephrenic and catatonic patients than among the children of simple and paranoid schizophrenics. The rates were 20.7, 21.6, 10.4 and 11.6 per cent for the children of hebephrenic, catatonic, simple and paranoid schizophrenics respectively. Analyzing the data by Schulz's method, he concluded that the findings were compatible with an hypothesis of biological homogeneity. Slater (19), however, analyzed the data differently and concluded that the clinical subtypes were genetically heterogeneous.
   b. Zehnder (21) compared schizophrenic siblings regarding their subtype diagnosis. She found equal proportions of sibships with similar or with dissimilar forms, and concluded in favor of biological homogeneity of schizophrenia. Otherwise, she questioned, why should dissimilar forms occur in sibships at all? Slater (19), however, reanalyzed Zehnder's data, found that the distribution of Kraepelinian subtypes in pairs of siblings showed a strong positive correlation, and attributed this similarity to the action of common modifying genes.
   c. Elsässer (4) found that the schizophrenic children of two schizophrenic parents with the same subtype diagnosis did not always belong to the same subtype as their parents. This led him to conclude that the Kraepelinian forms were genetically interchangeable.
4. *Genetic differentiation of typical from atypical schizophrenia.*
   a. Leonhard (8) found that his typical cases with deteriorative outcome showed much *less* apparent heredity than atypical cases which had a more variable course of illness and favorable prognosis.
   b. Elsässer (4) found that when both parents were schizophrenic, if the child had a psychosis at all it was likely to be schizophrenic. However, this was true primarily if the parents had a "typical" form of schizophrenia, suggesting that this type had a more direct hereditary effect with respect to schizophrenia than nontypical forms.

From this brief summary it can be seen that although the issues are of fundamental importance we are not in a position to draw conclusions with confidence about any one of them. Planansky concluded that twin studies were more promising as a method of attacking these problems than analyses of sibships and other family groupings.

In this study a novel method of analysis of a sample of twins has been used to approach the question of biological unity in schizophrenia.[2] The method involves only

[2] The rationale for this method has been stated most aptly by Allen (1) as follows. "It is likely that for many medical conditions the same clinical picture may be produced either by heredity or by

monozygotic twins. The twin pairs in the sample who are concordant with respect to the criterion, in this case schizophrenia, are compared with the pairs who are discordant. The comparisons made are dictated by specific hypotheses proposed by the investigator.

It was reasoned as follows. If there is a significant genetic factor in schizophrenia, it is especially likely to be represented among the one-egg twin pairs if both twins have the illness. Conversely, if there is a subgroup of schizophrenia which has either no hereditary basis or one which is only mildly contributory, this subgroup should be more likely to be represented in a group of one-egg twins in which only one twin has the illness.

Specifically, the hypotheses proposed were as follows.

1. Typical or process schizophrenia is found more frequently among concordant pairs, atypical or reactive schizophrenia occurring more often among discordant pairs of one-egg twins.

2. The severity of the illness is greater in concordant than in discordant twins.

3. History of schizophrenic illness is found more frequently in the families of concordant than of discordant twin pairs.

METHOD

Obtaining a well-selected sample of identical twins with at least one of the pair schizophrenic involves a tremendous output of time and effort. Such samples are therefore rare—there are in existence only five twin studies which have paid attention, however adequate, to problems of sampling: Luxenburger (9), Rosanoff *et al.* (16), Essen-Möller (5), Kallmann (7) and Slater (20). The yield of subjects in such studies is so small that it behooves us to extract as much information as we can from them.

Lacking the original data, one can make the proposed type of analysis only if the case histories are fairly detailed and if all, or almost all, the cases are published. Only the Essen-Möller (5) and Slater (20) studies meet these criteria. Unfortunately, in the Essen-Möller study there are only seven pairs of identical twins of whom at least one was said to be clearly schizophrenic, and these are too few for separate analysis. In the Slater sample there are 37 such monozygotic twin pairs, and their case histories are published in sufficient detail to make tests of the above hypotheses possible.

Slater's case histories provide the raw data for the present study.[3] Methodologically, there is a great advantage in testing the above hypotheses with data collected and published by another investigator who did not have these hypotheses in mind at the time of the original investigation. This virtually insures against the intrusion of bias which could unwittingly slant the collection or reporting of data either for or against these hypotheses. Unfortunately, such analyses are only occasionally possible, since the relevant data usually have not been obtained or published in the original report.

The cases were not simply dichotomized as process or reactive. Rather, a method was used which yielded a range of scores on a continuum related in most important respects to this dichotomy. Ratings were made from the histories of each twin's premorbid social and sexual adjustment, using the Phillips Scale of Premorbid Adjustment

environment. In such a case, a sample of single-born probands will contain a proportion of the environmental cases, or phenocopies, and a family study has to start out by lumping the relatives of both types of case. If, however, one starts with monozygotic twins as probands, a separation of the concordant and discordant pairs will in general segregate the genetic cases from the phenocopies. This permits from the outset a separate analysis of two groups of relatives practically without reference to familial incidence, so that the latter can be studied as an independent variable in the cases presumed to be genetic. Even with a very incomplete separation of genetic from non-genetic cases, the method may bring to light important statistical differences between the two groups of relatives. At the least, this method of analysis provides another type of internal control in twin studies."

[3] The author acknowledges gratefully Dr. Slater's spirit of scientific co-operation in making his case material available to interested investigators for study and evaluation.

TABLE 1

Phillips Premorbidity Scores of the Monozygotic Male Twins*

Concordant pairs			Discordant pairs		
Case #	Index case	Cotwin	Case #	Index case	Co-twin
30†	25	25	61†	22	22
80/174	27‡	24‡	133	20	9
87	24	21	202	24	16
240	25	28	231	22	15
287	20	23	17	21	15
			160†	19	19
Sum	121	121	Sum	128	96
$\bar{X}$	24.2	24.2	$\bar{X}$	21.3	16.0
S.D.	2.32		S.D.	1.60	4.00

* Phillips divided his scale so that subjects scoring 15 and above were called premorbid poors, 14 and below premorbid goods. Garmezy has felt that the middle range of scores, around 15, are less reliable prognosticators than more extreme scores.

† In Cases 30, 61, and 160, not enough information is provided to permit differentiation of the twins by these ratings.

‡ Since both twins are index cases, a coin was tossed to decide which twin to place in which column. This is done merely for presentational purposes and affects none of the conclusions.

(13). The scale yields a range of possible scores from 2 to 30; the higher the score, the poorer the patient's adjustment prior to his illness. At the lower end of the range of scores, subjects are called "premorbid goods," and at the higher end, "premorbid poors." The "goods" have a favorable prognosis, the "poors" unfavorable. In respect to prognosis as well as social and sexual history, the "poors" are representative of the group usually referred to as process, typical, nuclear, etc., while the "goods" are representative of patients ordinarily called reactive, atypical, peripheral, psychogenic, etc. Since the Phillips Scale has been validated for male subjects only, the rating could be made only for the male twins. The scale has been used with considerable success at Duke University in its program of research on the psychology of schizophrenia (15).

Reliability of the Phillips premorbidity ratings is found to be good even when social and sexual histories are only fragmentary. In order to check upon this a separate sample of case histories was obtained, about as detailed as Slater's. These were rated independently by two judges and the writer. One judge made Phillips premorbidity ratings on seven cases, and the other, a more sophisticated judge, made ratings on five cases. The average difference between the judges' ratings and mine was 2.7 and 2.0 scale points respectively.

As indices of the severity of illness, the patient's age at the time he was first admitted as schizophrenic to a clinic or hospital was used, as well as Slater's evaluation of the clinical condition of the patient at the time Slater examined him.

RESULTS

Table 1 presents the Phillips premorbidity ratings for each male twin in the sample.

Three main points brought out by Table 1 need to be considered.

1. Our first hypothesis leads us to predict that the premorbidity scores of the discordant index cases will be lower than the scores of the concordant index cases or the scores of all concordant twins combined. Although the mean premorbidity scores are rather high (poor) for both the concordant and the discordant schizophrenic twins, there is a slight difference of 2.9 scale points in favor of the discordant schizophrenic cases ($t = 2.531$, $P < .05$). Despite the fact that this finding accords with the prediction, there is not a single case among the discordant index cases which would be classified as a premorbid "good" (a score of 14 or less) by the Phillips Scale.

We can say that a relationship between premorbid adjustment and concordance or discordance among the monozygotic twin pairs appears to be indicated by the data. We are not able however to draw any inferences from these data about genetic differences between process and reactive schizophrenia since reactive types, as reflected

by good premorbid scores, do not occur in the sample at all. To test for such a difference, it may be necessary to find monozygotic twin index cases who are clearly of the reactive type and: a) see if their cotwins more often are free of schizophrenic illness than are the cotwins of process type cases; or b) see if the cotwins of both types of index cases are concordant with respect to these types.

2. Why is there an absence of reactive cases in the sample? Does reactive schizophrenia occur only rarely among one-egg male twins? There are no data available to suggest that its occurrence is less frequent among such twins than in the population generally. Unfortunately, we have no satisfactory information about what the proportion of reactive to process cases is in a population of newly hospitalized schizophrenic subjects. In any event, the sample seems to be biased in favor of process-type cases. One may wonder if such a bias may not creep into other twin studies when sampling is not even more rigorously controlled.

3. If we examine the discordant twin pairs, we see that in each instance where the twins could be differentiated on this scale, the twin who had the better premorbid social and sexual history was the one who escaped the illness. The scores of these twins tend to be in the middle range of the scale, except the cotwin in Case 133, whose low score is based on his statement that he had married and sired a child, though his sister doubted this. If we had disallowed his own report, his score would also have been in the middle range. Unfortunately, in two of the six pairs not enough information was available to differentiate the twins. Even so, a Mann-Whitney U test indicated that the two groups were reliably differentiated by the Phillips Scale ($U = 4.5$, $P = .017$). Thus, there is a suggestion that whether or not a twin among the discordant pairs developed schizophrenia seemed to be more closely associated with his premorbid history than with his genotype.

TABLE 2

Age of First Admission for Schizophrenia of the Affected Monozygotic Male Twins

Concordant pairs			Discordant pairs	
Case #	Index case	Cotwin	Case #	Index case
30	17	22	61	45
80/174	27*	28*	133	35
87	17	never hospitalized suicide, 28	202	38
240	27	29	231	29
287	25	26	17	40
			160	39
Sum	113	105	Sum	226
$\bar{X}$	22.6	26.25	$\bar{X}$	37.7
S.D.	4.29		S.D.	4.89

* Both twins were index cases. See footnote‡, Table 1.

Our second hypothesis is that the severity of the illness is greater in concordant than in discordant twins. More specifically, it was predicted that the disease would have an earlier "onset" and worse outcome for the concordant twins. Time of onset is notoriously difficult to pinpoint in any given case, but for purposes of group comparison, age at first admission for the illness is perhaps the best objective indicator of earliness or lateness of onset. Following the suggestion from Table 1 that the male concordant and discordant pairs may be genotypically different, we analyzed age at first admission and subsequent data separately for the male twins.

From Table 2, it is clear that the concordant male twins were hospitalized much earlier than the discordant male twins, the means being 24.2 years vs. 37.7 years. There is virtually no overlap of the two groups. The mean age difference is 13.5 years, which is significant at well beyond the .001 level.

The clinical condition of each patient at the time of examination is described by Slater in fair detail. Key phrases by Slater summarizing "outcome" of the illness at this point in time are quoted in Table 3.

TABLE 3

Clinical Outcomes of the Schizophrenic Illness for the Concordant and Discordant Monozygotic Male Twins

Concordant pairs			Discordant pairs	
Case #	Index case	Cotwin	Case #	Index case
80	"motionless lump"	"deteriorating"	61	"no evidence of gross affective dementia or . . . thought disorder."
80/174	"manneristic, suspicious" AWOL, stayed home	"deterioration steady and extreme"		
87	"deteriorated"	never seen by Slater	133	"well-preserved"
			202	"coherent; clearly orientated; memory preserved"
240	"malignant"			
	"incoherent, gross thought disorder"	"deteriorated"		
			231	"well-preserved"; discharged; working
287	"fairly normal"; married, working	"schizoid"; partly employed by father, at home	17	"remarkable preservation of personality"; discharged; not working
			160	"almost completely deteriorated"

If we describe these outcomes as simply "good" or "poor," the latter term covering deteriorative outcomes, the former nondeteriorative, six of the nine rateable concordant twins had a poor outcome as compared to one of the six discordant twins. Fisher's exact probability test yields a p-value of .078, which falls barely short of the usually accepted .05 criterion of significance.

It thus appears that the hypothesis of a more severe illness in the concordant than discordant male twins has considerable support in these data. Because of these differences, it seemed worth inquiring into the Kraepelinian subtype diagnoses of all the affected twins, insofar as such diagnoses could be estimated.

Slater reports that in all concordant uniovular twins, male and female, only one twin was without catatonic signs and symptoms, and it appears from Table 4 that the concordant male twins were predominantly in the catatonic subgroup while the discordant male twins were primarily paranoid.[4]

Our third hypothesis led us to predict that we would find a higher incidence of schizoid-schizophrenic illness in the families of the concordant than of the discordant pairs.

According to Table 5, four of the five families of the concordant pairs have a markedly schizoid member, or a member whose illness is probably schizophrenic, but no such cases are found in the families of the discordant pairs. The nephew of Case #17 had been admitted to a psychiatric clinic at age 18 with paranoid schizophrenia, but Slater states that some clinical signs suggested pituitary abnormality from which the patient had recovered, and he had remained well for the 25 years since. The higher incidence among the families of the concordant twins cannot be due to a greater number of relatives in these families, since there were 24 siblings of the discordant pairs

[4] Of course, these are mostly chronically ill patients who do not represent the entire range of catatonic and paranoid subtypes. Clearly, there are acute and intermittent catatonics whose course is generally more favorable than that found in the concordant twins, as well as a fairly large group of acute paranoid patients whose course leads to deterioration. Neither hebephrenic nor simple schizophrenia is represented in this group of subjects. A larger sample may have included a wider range of subtypes.

TABLE 4

*Suggested Subtype Diagnoses of the Concordant and Discordant Monozygotic Male Twins**

Concordant pairs			Discordant pairs	
Case #	Index case	Cotwin	Case #	Index case
30	(S) Catatonic	Mixed, catatonic and paranoid	61	Paranoid, with depressive features
80/174	Mixed, predominantly catatonic with paranoid features		133	(S) Paranoid, or, schizo-affective with manic-depressive type mood swings.
87	(S) Catatonic	Not diagnosable		
240	(S) "Mainly catatonic"		202	Paranoid
287	Paranoid, with catatonic features		231	(S) Paranoid
			17	(S) Paranoid
			160	Predominantly paranoid initially, before deterioration

* Where Slater had indicated a diagnosis made by the hospital at admission or reevaluation, or by himself based on his own examination, that diagnosis has been preceded by the symbol (S). The remaining diagnoses are suggested by the author as a best estimate, based on the descriptions provided.

TABLE 5

Incidence of Schizophrenic Illness in the Families of the Monozygotic Male Twins

Case #	Concordant pairs	Case #	Discordant pairs
30	Sister, markedly schizoid, socially withdrawn	61	None
80/174	Mother shared some of son's delusions	133	None
87	Maternal aunt died in mental hospital	202	None
240	Grandfather, "religious mania," in mental hospital till death	231	None
287	None	17	Nephew, once diagnosed as paranoid schizophrenic, but called "pituitary disturbance" by Slater.
		160	None

who survived to a susceptible age as compared to only 11 such siblings of the concordant pairs. Other kinds of psychiatric disturbances occurred with high frequency in the families of both concordant and discordant pairs. These included mental deficiency, alcoholism, psychopathy, neuroses such as anxious-phobic states and reactive depression, and personality problems such as diffidence, excessive worrying, etc.

Although we cannot make premorbidity ratings for the female twins, we can test hypotheses 2 and 3 with these cases. In this regard, the first point of note is that whereas there were only 11 pairs of monozygotic male twins in the sample, there were 26 pairs of female twins. Of the 11 male pairs, five were concordant. Of the 26 female pairs, 19 were concordant. These differences are striking and raise the question of whether we are dealing here with sampling errors or real differences. Actually, there is a large variety of studies which contain a higher number of female index cases and higher concordance rates among females with regard to mental illness. It would take us too far afield to review these studies here, but they appear to throw additional light on the question of hereditary vs. life-experience factors in schizophrenia.

Since the N is so large, we will not set up tables for the female pairs as we did for the males. We may summarize the findings briefly by stating that the hypothesis of

TABLE 6

Incidence of Schizophrenic Illness in Families of the Concordant and Discordant Monozygotic Pairs: Both Sexes

	Family history positive for probable schizophrenic illness	Family history negative for probable schizophrenic illness
All concordant pairs	13	9
All discordant pairs	1	12

greater severity of illness in the concordant than in the discordant twins finds no support in the data, whereas the hypothesis of higher incidence of schizophrenia in the families of the concordant pairs finds additional support. The mean age at first hospitalization was 31.72 years for the concordant female twins, 33.14 years for the discordant female twins. The difference is not noteworthy. With respect to outcome, 14 of 34 rateable concordant female twins were said to be deteriorated as compared to four of the six rateable discordant female twins. The difference is not statistically significant but is actually in the opposite direction from that predicted.

These findings reinforce opinions stated in the past that there are striking differences in the clinical conditions of male and female schizophrenic patients, and that females are less predictable. Similarly, whereas a premorbidity scale to predict outcome of illness in schizophrenic males could be constructed fairly easily, one has yet to be worked out which will as reliably predict outcomes with schizophrenic females. Thus, when dealing with clinical phenomena, it is judicious not to pool the sexes.

Of the 19 concordant female pairs, histories of probably schizophrenic illness occurred in nine families, absence of such history occurred in eight families, and in two families there was sufficient doubt about a case to defer a definite classification. Of the seven discordant female pairs, absence of schizophrenic history was noted in six families. In the seventh family, one relative was said to have had a schizophrenic episode at age 19, recovered in short order, and to have been well since. If we pool the family incidence figures for both sexes, we find the distribution shown in Table 6.

It can be seen from Table 6 that the families of the discordant pairs are virtually free of schizophrenic illness. In the one case where the illness is said to have occurred, the course was unusually rapid and benign. By contrast, about 60 per cent of the families of the concordant pairs showed evidence of a schizophrenic illness. Testing the significance of the difference, we find a chi-square, corrected for continuity, of 6.98, which is significant at well beyond the .01 level.

DISCUSSION

Our analysis provides support for the hypothesis of biological heterogeneity in schizophrenia. On the one hand, the combined findings of discordance in monozygotic twins and the virtual absence of schizophrenia among members of their families are strongly suggestive that a schizophrenic gene is not the responsible agent in the illness of these twin index cases. For the male twins we may specify even further that the illness is more closely linked to the type of premorbid social and sexual history that the twin experienced than to his genotype. We may state too that the type of illness in males in which genetic factors are of minimal or no importance is one which has a relatively late onset and benign outcome in that the illness does not progress to deterioration and is manifested clinically as a predominantly paranoid syndrome, sometimes accompanied by prominent affective features.

On the other hand, the combined findings of concordance in the monozygotic twins plus the fact that history of probable schizophrenic illness occurs in approximately 60 per cent of their families is strongly suggestive of an hereditary determinant. Of the

male twins we may add that genotypic expression is manifest early in that these patients have both a poorer premorbid social and sexual history and a relatively early onset of the disease. Moreover, the illness tends to be malignant, with a preponderance of catatonic symptoms.

Evidence of biological heterogeneity with respect to the paranoid and catatonic subtypes can be found in the data of Kallmann (6) and Zehnder (21), as pointed out by Slater (19). Our findings suggest that the relationships found in these studies could be made clearer still if the cases were analyzed separately by sex, and if, among the male paranoid schizophrenics, those who had a relatively late onset and benign outcome were separated from the other patients included in this subtype.

There is also evidence, in keeping with our findings, that genetic factors play a differential role with respect to course and outcome of the disease. Slater (19), analyzing the sibling pairs of Zehnder, who had classified their outcomes as "dementia, chronicity, or remission," found a positive relationship between brothers and sisters in this respect. Slater (20) grouped together all concordant uniovular twins, binovular twins, and sibling pairs, and also concluded that genetic factors helped to determine whether the illness took a relapsing form or not. Luxenburger (10) had already noted a tendency for the end-state of schizophrenic illness in concordant monozygotic twins to correspond, especially in the hebephrenic and catatonic forms of the disease as compared to the paranoid or mixed forms.

Some inconclusive evidence also exists to show that concordance with respect to age of onset of schizophrenia is greater when the hereditary relationship is closer. Thus, Luxenburger (11) has presented tables which, analyzed statistically, show that uniovular twins have a more similar age of onset than siblings, and siblings a more similar age of onset than a selected sample of unrelated cases. Slater (20) also found a higher correlation between concordant one-egg twins with respect to age of onset than between concordant sibs, although for two-egg twins the correlation was higher than for one-egg twins.

The latter apparent contradiction suggests that the contribution of non-hereditary factors to an aspect of the illness such as age of onset may be considerable. Neither can such factors be ruled out with respect to clinical subtype and outcome of the illness, but the case for genetic determinants in these respects is somewhat stronger.

Two matters of moment which must be considered even more seriously are sampling procedures in twin studies of psychiatric illness and male-female differences with respect to such illnesses. Each of these problems merits separate review in a manner too detailed to attempt here.

In a recent provocative article, Anastasi (2) has pointed out that the heredity-environment controversy is far from dead, although some scientists may have wished long since to bury it. Rather, she has suggested, if we ask the right questions, and are skillful enough in applying techniques of study and analysis, our body of knowledge about many fundamental matters may be enriched considerably. It is in this spirit that the present study has been undertaken.

SUMMARY AND CONCLUSIONS

This study addresses itself to the question of biological unity in schizophrenia. The case histories of monozygotic twins published by Slater (20) are analyzed with respect to three hypotheses. There is almost a total absence of schizophrenia in the families of the discordant twin pairs, but the illness occurs in about 60 per cent of the families of the concordant twin pairs. The affected discordant male twins tend to have a later age of onset and more favorable outcome than the concordant male twins. The former tend to be of the paranoid subtype, the latter catatonic. Of the discordant male twins, the one who does not become ill tends

to have the better premorbid social and sexual history, and the twin who does become ill tends to have a better premorbid adjustment than do the concordant twins. It is concluded that, biologically speaking, at least two broad groups of schizophrenia are differentiated by this method of analysis: in one, the genetic contribution is absent or minimal; in the other, the genetic contribution is probably considerable.

REFERENCES

1. Allen, G. Discussion, in Proceedings of the Conference on Problems and Methods in Human Genetics. Amer. J. Hum. Genet., **6**: 162–164, 1954.
2. Anastasi, Anne. Heredity, environment, and the question "how?" Psychol. Rev., **65**: 197–208, 1958.
3. Bleuler, M. Vererbungsprobleme bei Schizophrenen. Z. Neur Psychiat., **127**: 321–388, 1930.
4. Elsässer, G. *Die Nachkommen Geisteskranker Elternpaare*. Thieme, Stuttgart, 1952.
5. Essen-Möller, E. *Psychiatrische Untersuchungen an einer Serie von Zwillingen*. Munksgaard, Copenhagen, 1941.
6. Kallmann, F. J. *The Genetics of Schizophrenia*. Augustin, New York, 1938.
7. Kallmann, F. J. The genetics of psychoses: An analysis of 1,232 twin index families. In *Congrès International de Psychiatrie, Rapports VI Psychiatrie Sociale*. 1–27, Paris, 1950.
8. Leonhard, K. *Die Defekt—Schizophrenen Krankheitsbilder*. Thieme, Leipzig, 1936.
9. Luxenburger, H. Vorläufiger Bericht über psychiatrische Serienuntersuchungen an Zwillingen. Zeitschr. ges. Neurol. Psychiat., **116**: 297–326, 1928.
10. Luxenburger, H. Psychiatrische-neurologische Zwillingspathologie. Zbl. ges. Neurol. Psychiat., **56**: 145–180, 1930.
11. Luxenburger, H. Untersuchungen an schizophrenen Zwillingen und ihren Geschwistern zur Prüfung der Realität von Manifestationsschwankungen. Zeitschr. ges. Neurol. Psychiat., **154**: 351–394, 1935.
12. Penrose, L. S. Survey of cases of familial mental illness. Mimeographed ms., abstracted in Digest Neurol. Psychiat., **13**: 644, 1945.
13. Phillips, L. Case history data and prognosis in schizophrenia. J. nerv. ment. Dis., **117**: 515–525, 1953.
14. Planansky, K. Heredity in schizophrenia. J. nerv. ment. Dis., **122**: 121–142, 1955.
15. Rodnick, E. H. and Garmezy, N. An experimental approach to the study of motivation in schizophrenia. In *Nebraska Symposium on Motivation*, pp. 109–184, U. of Neb. Press, Lincoln, 1957.
16. Rosanoff, A. J., Handy, L. M., Plesset, I. R. and Brush, S. The etiology of so-called schizophrenic psychoses. Amer. J. Psychiat., **91**: 247–286, 1934–35.
17. Rüdin, E. *Zur Vererbung und Neuentstehung der Dementia Praecox*. Springer, Berlin, 1916.
18. Schulz, B. Empirische Untersuchungen über die Bedeutung beidseitiger Belastung mit endogenen Psychosen. Zeitschr. ges. Neurol. Psychiat., **165**: 97–108, 1939.
19. Slater, E. Genetical causes of schizophrenic symptoms. Mschr. Psychiat. Neurol., **113**: 50–58, 1947.
20. Slater, E., with the assistance of James Shields. *Psychotic and Neurotic Illnesses in Twins*. Her Majesty's Stationery Office, London, 1953.
21. Zehnder, M. Über Krankheitsbild und Krankheitsverlauf bei schizophrenen Geschwistern. Mschr. Psychiat. Neurol., **103**: 231–277, 1941.

A METHODOLOGICAL STUDY OF FREUDIAN THEORY: I. BASIC CONCEPTS

ABRAM KARDINER, M.D.[1] AARON KARUSH, M.D.[1] AND LIONEL OVESEY, M.D.[1]

The purpose of this paper is to subject the assumptions upon which Freudian theory is based to a critical examination. We will examine psychoanalytic methodology and appraise the reasoning and the operational devices used by Freud to explain his clinical observations. In so doing we will try to make clear which of Freud's concepts are acceptable to us today. We will then demonstrate the methodological inconsistencies and theoretical defects which in our opinion seriously impair the effective application of psychoanalytic theory to research and therapy. Furthermore, wherever possible, we will suggest alternative explanations for those hypotheses that we feel are defective. Our explanations will be derived from an adaptational frame of reference rather than the Freudian instinctual one. The difference between the two theoretical frameworks will be clarified in the body of the paper, which will be divided into four parts. The first will consider the basic psychological concepts that grew out of Freud's early investigations into the causation of neuroses and the nature of dreams. In this section we will review the methodological principles used by Freud to derive these concepts from his clinical data. The second will consist of a discussion of the libido theory, the central role assigned to the oedipus complex, and the derivation of character traits. The third will consider the theory of narcissism, the concept of constitutional bisexuality, and the dual instinct theory. Finally, the fourth part will deal with the new structural hypothesis, the revised theory of anxiety, and post-Freudian ego psychology. We will not take up every minute detail of Freudian theory, but instead will concentrate on the major constellations and their revisions in the historical sequence of their development. We will omit consideration of Freudian sociology, which has been critically appraised elsewhere by Kardiner (7, 8), and we will not at this time discuss Freud's theories about the technique of psychoanalytic treatment.

In our evaluation of Freud's discoveries we will take the position that psychoanalysis is an empirical discipline. We will, therefore, use an operational critique based upon propositions that can be subjected to clinical observation. We shall try to avoid purely philosophical issues about the "essence" or "nature" of things. No psychology today has the tools to investigate the meaning of life itself. Psychoanalysis is a method of defining relationships among psychological phenomena. It operates on data with the aid of certain hypotheses and logical assumptions which are either semantically clear or not. Some of these operations enhance our knowledge, others are either erroneous or end in tautologies. Explanations that restate our assumptions in different words pro-

[1] The Psychoanalytic Clinic for Training and Research, Department of Psychiatry, College of Physicians and Surgeons, Columbia University, New York, N. Y. This is the first section of a four-part article by these authors, to appear in successive issues of JNMD, in which Freudian theoretical assumptions are subjected to critical examination. The present paper considers the basic psychological concepts that grew out of Freud's early investigations into the causation of neuroses and the nature of dreams, and reviews the methodological principles used by Freud to derive these concepts from his clinical data. The second paper consists of a discussion of the libido theory, the central role assigned to the oedipus complex, and the derivation of character traits. The third section considers the theory of narcissism, the concept of constitutional bisexuality, and the dual instinct theory. The fourth paper deals with the new structural hypothesis, the revised theory of anxiety, and post-Freudian ego psychology.

vide no new information and, unless they improve conceptualization, have no scientific usefulness. In this paper we will attempt to differentiate those Freudian hypotheses that augment our knowledge from those which, in our opinion, do not.

Let us consider more closely the standards we believe ought to be applied rigorously to the construction of a psychoanalytic theory. As we have just stated, psychological knowledge represents a union of observable phenomena, or data, with explanatory assumptions which establish relationships between these data. Scientifically useful assumptions, however, must fulfill an additional requirement. They must be conceived in a conceptual framework which points the way to new observations that can verify or refute the original assumption. An assumption, however, may be structured in such a way as to be incapable of verification or refutation by new data. No matter how attractive such an assumption may seem, it cannot, therefore, add to our knowledge. The operational value of a useful assumption can best be demonstrated through an example. For this purpose we would like to cite the work of Levy on finger-sucking in infants (10). He observed that after feeding was over and hunger was satiated, some infants sucked their fingers, while others did not. His assumption was that a sucking need existed which was independent of the need for food to relieve hunger. Those infants, therefore, who suffered a sucking-time deficiency during feeding had to relieve themselves by subsequent finger-sucking. He then put this hypothesis to an experimental test. He measured the sucking-time spent by a large number of infants at each feeding and correlated these observations with finger-sucking. He found that infants who sucked the longest at feedings seldom sucked their fingers after the meal was over, while those who sucked the least at feedings were precisely the ones who later engaged in finger-sucking. Thus, the original assumption led to the discovery of new data about variability in sucking-time and its subsequent effects. The new data, in turn, validated the original assumption, and hence established its scientific usefulness. It is important to grasp, however, that had the experiment failed, and the original assumption been refuted, it would still have been a scientifically useful assumption. It would still have added to our knowledge, but in the negative sense of refutation, rather than in the positive sense of validation. The observer would then have been forced to discard the assumption and to seek out a new one.

It is impossible to start reasoning about either psychological or physical organismic processes without assumptions. The physical sciences rest upon a hard core of physical reality that is easily identified and universally accepted, but psychological reality, unfortunately, has no fixed system of recognizable entities and symbols to guide our assumptions. As a result, psychological assumptions often have different meanings to different observers and, worse, may be called upon to substitute for unavailable data. Even the entities we would like to call objective data are often disputed. For example, two psychiatrists may observe an affect in the same patient and yet disagree on its quality or intensity. The immediate task that confronts all of us, regardless of our theoretical orientation, is to agree on those observable entities which constitute the "hard core" of psychological reality. Such agreement would do much to facilitate the necessary revision of faulty hypotheses.

The subject matter of psychoanalysis should include the adaptive processes of the individual from birth to death. These processes can be described only in terms of motivation, intrapsychic mechanisms, learned responses, and the developmental factors that influence each of these. A psychoanalytic theory, therefore, must be capable of tracing ontogenesis from infancy to maturity and differentiating effectual from ineffectual adaptation. It must also distin-

guish between learned and inborn patterns of behavior while, at the same time, it keeps track of the relations between the two.

The common paradigm for several psychologies, including psychoanalysis, is the reflex arc, wherein a stimulus produces an immediate response by virtue of a built-in system that connects perception with the executive apparatus. Freud added several dimensions to this simple formulation and opened up new sources of data which made possible new assumptions about the psychological events that intervened between the stimulus and the response. The new data were derived from current and past life experiences, dreams, free association of ideas, and seemingly innocent errors such as slips of the tongue. It is these data which constitute the empirical basis of psychoanalysis. From them Freud drew conclusions which embodied the following general assumptions: 1) Adult behavior was rooted in childhood experiences; 2) Frustration of sexual needs led to neurosis; 3) Much of behavior was integrated by unconscious mental processes. These assumptions were common sense generalizations that stayed very close to the data and followed the laws of logic. They established relationships between hitherto isolated phenomena and were strictly operational in their purpose, that is, they provided Freud with useful clinical guides for understanding his patient's problems. They constituted new psychological knowledge because they were capable of validation or refutation through clinical investigation. For the most part, all three have been repeatedly validated and, as we shall see, have led to vast stores of new data and new hypotheses. Their general acceptance today is tacit recognition that their scientific worth has been well established.

The same, however, cannot always be said for the libido theory or its many revisions. This theory took its origin from the earlier generalization which related neurosis to sexual frustration. The theory contained a number of assumptions which, unlike those of the first order, were much farther removed from the available data. Furthermore, many of the new assumptions were no longer common sense attempts to establish logical relationships, but became instead philosophical speculations into the "nature" of things. These speculations could not be supported by the psychological data themselves and Freud filled the gaps with constructs borrowed from nineteenth century biology, physics, and chemistry. The outcome was a theory built about the following second order of assumptions. 1) The sexual instinct was the prime mover in all behavior, both normal and neurotic; 2) The sexual instinct had an energic component, the libido, which, like physical energy, sought outlets for release; 3) The sexual instinct had a phylogenetic history and a constitutionally-predetermined course of development.

We are not, for the moment, primarily concerned with the validity of any of these assumptions. At this point we wish only to discuss the issue of scientific usefulness. The assumptions of the libido theory have many aspects, some of which satisfy the criteria we have set down for psychological knowledge, while others do not. Thus, the primary role in human behavior of the sexual instinct and its constitutional predetermination can be tested clinically. The concepts of instinct and instinctual energy, however, as used by Freud, often do not lend themselves to any type of scientific exploration. In many respects we feel they can be neither validated nor refuted, nor do they help us unearth new data. The analysis of Freudian theory which follows in this paper, therefore, will pay particular attention to three general subjects: 1) the nature, origin, and consequence of those assumptions in Freudian theory which, by our standards do not add to psychological knowledge; 2) the identification of those assumptions made by Freud which remain scientifically useful; 3) the formulation of new assumptions

which we believe will add to psychological knowledge.

The first psychoanalytic theory proposed by Freud grew out of the clinical discoveries of Charcot (3) and Breuer (1). Charcot had demonstrated that the hypnotic trance was an artificial hysteria. Ideas, memories, and impulses in both conditions lost their connections with one another and existed in a state of *dissociation.* Breuer introduced a modification of hypnotic technique in the treatment of hysteria. This modification occurred in a rather unexpected way, owing largely to the temperament of Josef Breuer, who could sit patiently and listen to the outpourings of Anna O. during her lapses into autohypnosis. By permitting her to talk and *abreact,* he transformed hypnosis from a purely command-submission procedure into a more interactional experience between himself and the patient which was protective, forgiving, and permissive. This technical innovation was important because it created the opportunity to examine, in slow motion, the phenomenon of dissociation and to study those experiences of the patient from which it arose. The practice of hypnosis, however, made an examination of the mental mechanisms that produced dissociation quite impossible since these mechanisms were circumvented by the hypnotic command. The problem was solved when Freud omitted hypnosis altogether and substituted free association in a fully conscious state. He, thus, made it possible to observe not only the content of dissociated states, but also the mental mechanisms that produced them.

The use of free association permitted a more meticulous examination of content and exposed the psychic forces that governed the contiguity of ideas. Freud discovered that the abreaction or discharge of affects was blocked by *resistance* which interfered with the free flow of ideas. It was this resistance that caused the affects to be dissociated from memories in the sense that they were denied access to awareness and to avenues of discharge and thereby led to the symptoms of which the patient complained. The closure of avenues of discharge, association, or expression, therefore, became the first psychodynamic principle. The memories that emerged through free association revealed that the dissociated state was the end result of a process initiated by a traumatic event. It had already been demonstrated by Charcot that a physical shock or trauma could induce the dissociation that gave rise to a traumatic neurosis. Breuer and Freud believed that a psychic trauma could cause an analogous dissociation in hysteria by awakening unacceptable impulses which were accompanied by distressing affects. These affects were described by Freud as "fright, anxiety, shame, or physical pain" (1, p. 6). The relegation of the forbidden ideas to the unconscious (*repression*) prevented the appropriate discharge of the associated affects which then sought anomalous discharge through neurotic symptom-formation. Not until the repressed memories were made conscious could the impounded affects be abreacted in their original form. Only then would the patient lose his symptoms.

The basic idea of closure of avenues of discharge was thus explored from three points of view:

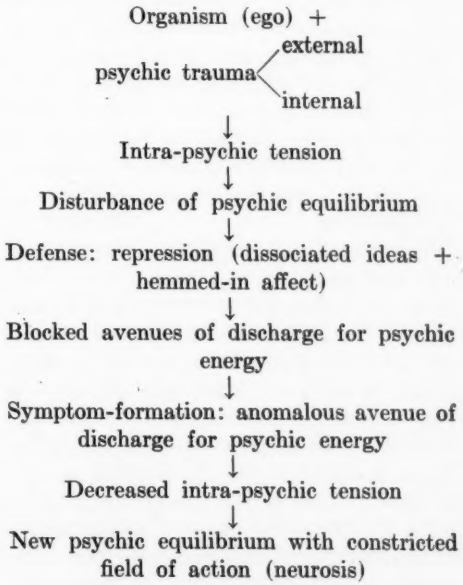
1. Content and its Alteration
   a. *Ideas* were denied associative connection and awareness.
   b. *Affects* were blocked from discharge.
   c. *Impulses* to action were inhibited.
2. Method of Alteration
   The alteration of content was the result of a mental mechanism, *repression.*
3. End Product of Alteration
   Repression produced dissociation, now expressed by a topological concept, *unconscious.*

Freud quickly realized that these alterations were protective in intent. They were defenses against impulses to gain forbidden pleasures. The need for defense, therefore,

supplied the motivation for repression, which prevented action dangerous to the patient by blocking awareness of the motivating ideas and impulses. Its function was to enhance security by constricting the effective field of action. The impairment that resulted from this inhibition thereafter became a permanent part of the executive apparatus and found its ultimate expression in symptom-formation.

Repression accounted for the process of alteration that gave rise to the hysterical symptoms, but not for the form they took nor their intensity. To explain the latter, Breuer and Freud drew on the teachings of Fechner (2), who believed that all natural phenomena were explicable in terms of physical and chemical forces. His concept of physical energy was borrowed by Breuer and Freud and applied directly to mental activity as "psychic energy" which, like its physical counterpart, was governed by the second law of thermodynamics. This enabled Freud to state, "There is a tendency to keep intracerebral excitation constant" (1, p. 197). This principle of constancy lay the foundation for the hypothesis that the choice of symptom and its intensity were determined by quantitative changes in the level of psychic energy. The investment of mental content by a quantity of psychic energy, that is, an energic charge, was given the name Besetzung, later translated into Greek as *cathexis*. It now became possible to explain the somatic manifestations of hysteria as transformations or *conversions* of psychic energy in which the physical symptom, through a shift in cathexis, became the symbolic substitute for the repressed idea. The energic explanation of conversion hysteria was later extended to symptom-formation in other disorders. Thus, for example, reaction-formation in obsessive-compulsive neurosis and projection in paranoia also represented shifts in cathexes which resulted in the altered ideation characteristic of these conditions.

Freud now had an operational frame of reference by means of which he could describe and explain psychopathology as a series of events that began with a disruption of psychic equilibrium by a trauma and ended in the restoration of the equilibrium through repression and symptom-formation. Once Freud recognized that a sexual impulse denied expression at puberty could act like a trauma by "increasing excitability and reducing resistances everywhere" (1, p. 244) he had a twofold source for the disturbance in equilibrium, an external one and an internal one, both resulting in increased intra-psychic tension. The operational frame of reference used in "Studies in Hysteria" (1) can be diagrammatically represented as follows:


Organism (ego) +
psychic trauma
external
internal
Intra-psychic tension
Disturbance of psychic equilibrium
Defense: repression (dissociated ideas + hemmed-in affect)
Blocked avenues of discharge for psychic energy
Symptom-formation: anomalous avenue of discharge for psychic energy
Decreased intra-psychic tension
New psychic equilibrium with constricted field of action (neurosis)

The new psychic equilibrium, however, was more apparent than real. The symptom did *not* restore the original state of equilibrium; it merely attempted to do so. It failed because it left the patient with a new—although less painful—disturbance of function. In that sense, the symptom was a lesser evil which substituted one disequilibrium for another. The restoration of the original

state of equilibrium could only be effected by undoing repression, restoring ideas to associative freedom, and releasing pinched-off affects. Hence, catharsis.

This was the earliest formulation of an adaptational maneuver, even though it was partly couched in energic terms. Implied within the frame of reference was a coordinating central agency (the ego) which altered perceptions of motivating impulses and removed them from effective influence by repression. The psychic trauma that set this maneuver in motion appeared to be sexual in nature. Freud's early patients related that as young children they had undergone seduction by nursemaids, governesses, domestic servants, tutors, teachers, or by other children. It seemed to Freud that the specific etiological factor in psychoneurosis was this infantile sexual trauma. He, therefore, concluded that the predisposition to psychoneurosis was caused by an *accidental external* influence on the sexual life of the child which perpetuated an unconscious wish for the forbidden infantile sexual gratification.

It is in "The Interpretation of Dreams" (4) that Freud applied this frame of reference to a different set of phenomena and validated its worth as an operational tool. He established that the dominant motivation in dreams was wish-fulfillment, while resistance and censorship were homeostatic devices whose purpose was to keep excitation at a constant level. He believed that the dream-wish was exactly the same forbidden infantile sexual wish that lay at the bottom of neurotic symptom-formation. Free association furnished him with the means for comparing events and images in the dream with the life experiences that gave rise to them. Comparisons between the two series showed that dream images were created by subjecting reality to certain alterations in the interests of wish-fulfillment. The processes of alteration comprised what Freud called the "dream work" and determined the representability of the latent dream-wish in the manifest dream-content. These processes were the identical mental mechanisms of defense which he later described more fully in his clinical studies of the neuroses: repression, regression, displacement, condensation, projection, denial, isolation, and reaction-formation. This discovery in "The Interpretation of Dreams" enabled Freud to conclude that dream-formation was identical with symptom-formation.

It is important to understand that the mental mechanisms were inferences drawn from observations of aberrant behavior induced by unacceptable motivations. They, therefore, represented the person's adaptive devices for coping with a conflict situation. Freud saw that the ego of the dreamer was in constant contact not only with external reality, from which it drew its cues, but also with its own past experiences. Thus, the adaptive response consisted of a modification of the primitive infantile wish in accordance with the necessities of logic and security, *i.e.*, "secondary process".

Freud was not content with these formulations, but sought a neurophysiological explanation for the transformation of unconscious ideas into sensory images. Here, again, he used the paradigm of the reflex arc, which he modified by introducing mnemic systems between the sensory stimulus and the motor response. He assumed that in general psychic processes, as did physiological ones, advanced progressively from the perceptual end to the motor end. The reverse seemed to take place in dreams. The motive force—the dream-wish—originated in the unconscious, but its energic charge, instead of being transmitted toward the motor end of the apparatus, moved in a backward direction toward the sensory end until it finally cathected the perceptual system. In this way it produced the hallucinatory revival of the perceptual images characteristic of the dream. The movement of excitation in dreams, therefore, was regressive, or as stated by Freud, "In regression the fabric of the dream thoughts is resolved

into its raw material" (4, vol. 5, p. 543). This energic hypothesis is the most perishable part of Freud's theory of dreams. It is subject to the same criticism that we shall make later of energic concepts in general.

The accumulation of clinical data eventually led Freud to abandon his environmental theory of neurosis in favor of a constitutional one. He discovered that many of the scenes of seduction reported by neurotic patients were in reality fabricated memories. These fantasies were projections that defended the child from the memory of his own sexual wishes and actions. Freud then discovered that normal persons reported the same infantile sexual experiences, both real and imagined.[2] He therefore concluded that infantile sexuality was a universal phenomenon that predisposed to neurosis only if the experience was traumatic and its remembrance repressed. This conclusion had far-reaching consequences for the future direction of psychoanalytic theory. It is best summarized in Freud's own words.

> "Investigation into the mental life of normal persons then yielded the unexpected discovery that their infantile history in regard to sexual matters was not necessarily different in essentials from that of the neurotic, and that seduction particularly had played the same part in it; the result was that the accidental influences receded still further into the background in favour of the influence of 'repression,' as I had begun to call what I had formerly termed 'defense.' The important thing, therefore, was evidently not the sexual stimulation that the person had experienced during childhood; what mattered was, above all, how he had reacted to these experiences, whether he had responded to them with 'repression' or not. . . ." (5)

[2] The sources of this discovery are not documented by Freud. We can only conjecture that this observation about normal development derived from his self-analysis.

At this point Freud made the crucial assumption that repression was determined by an inborn sexual constitution which underwent a fixed course of development. The disposition to neurosis now lay in the constitution, rather than in the environmental trauma. This shift in his thinking made necessary the study of the anatomic and functional evolution of the sexual constitution from infancy to adulthood. Freud's first theory of sexual ontogenesis was contained in "Three Contributions to the Theory of Sex" (6). This work marked the beginning of the libido theory.

Let us now examine Freud's operational tools more closely. Freud believed that mental processes could be fully understood only if they were described from three points of view: dynamic, topographic, and economic. Such a description comprised what he eventually called his "metapsychology." *Dynamic* referred to the interplay of mental mechanisms by means of which a need ultimately led either to action or to inhibition of action. These mental mechanisms were so distinctive and so easily verifiable that they have become the basic armamentarium of psychoanalytic theory. *Topographic* referred to the "functional" areas of the mind —conscious, preconscious, and unconscious —in which the conflict between motivational and countermotivational ideas took place. This division made it possible to differentiate those ideas that were subject to recall by voluntary means from those ideas that were repressed and hence could not voluntarily be recalled. *Economic* referred to the source, distribution, transformation and mode of discharge of psychic energy. This early hypothesis of psychic energy was later amended and vastly expanded by Freud in the libido theory.

The principles first established by Freud as "dynamic" and "topographic" are the fundamental principles of Freudian psychology and remain an integral part of modern psychoanalytic theory, both classical and adaptational. They may be sum-

marized as follows:

1. *Motivation.* In general, behavior is purposive and goal-directed.
2. *Pain-Pleasure Principle.* In general, behavior is designed to avoid pain and gain pleasure.
3. *The Unconscious.* Behavior is motivated to a great extent by ideas that exist without awareness.
4. *Psychodynamics.* Neurotic behavior is the resultant of conflicting motivational and countermotivational ideas.
5. *Principle of Defense.* Symptom-formation is defensive in intent. Repression is the pivotal mental mechanism of defense.
6. *Ontogenesis.* Childhood experiences exercise a vital influence on adult behavior and are repeated in the transference during the therapeutic process.
7. *Continuity of Behavior.* Any single item of experience can be fully understood only in the light of the total experience of a person. The past, present, and future are tied together in a unified whole. Whatever happens to a person in the present is related to causes in his past and to the motivational goals he hopes to achieve in the future.

We have omitted the concept of psychic energy from this list of fundamental principles despite the significance given it by Freud. This concept is still adhered to by classical theorists today, but in our opinion it is an assumption which adds nothing to our knowledge and hence should be discarded. Freud's description of mental activity as a manifestation of psychic energy was derived from known neurological phenomena. The process of sense perception evoked ideas and emotions which in turn produced behavior, normal and abnormal. Freud recognized the etiological significance of ideas and emotions, but he felt it necessary to interpret all psychological events in terms of physical forces. He simply substituted a hypothetical psychic excitation for the measurable energy of nerve conduction. This enabled him to set up parallel systems of describing behavior, not only psychodynamically, but also economically.

There is no neural basis for Freud's concept of psychic energy. The major objections to such a construct have recently been stated with great clarity by Lashley (9):

> "The concept of energy, as used in the physical sciences, is defined in terms of mass and acceleration... The various forms of energy (heat, electricity, sound, gravitation, etc.) are mutually translatable and are described in terms of structure or organization of more elementary mass movements (oscillation of electrons, *e.g.*). The characteristics or laws of each form of energy are rigidly determined by the structure, just as the mode of energy transformation of a gas engine is determined by the arrangement of piston and crankshaft.... The nervous system is such a limiting structure. Neural activity has been sufficiently well explored to rule out such broad assumptions as of the energy of the libido or of the id. Summation, potentiation, irradiation, and inhibition are fairly well, though not completely, understood. The energy of the nervous system is that of transmitted excitations, with its implied limitations and specificities. Energy dissociated from this, as postulated in field theories, is ruled out by definite experimental evidence. In particular, the derivation of psychic energy from one or a few 'instincts' finds no support in the nature of neural activity.... Where instinctive activities have been analyzed experimentally, as in our studies of hunger, mating and maternal behavior, there is nothing that suggests free or transferable energy. There are variations in thresholds of activity with consequent differences in the arousing stimuli, dominance of a reaction system with suppression of other activities, and changes in the effective stimulus and the pattern of response due to observable formation of associations. The

behavior can be explained without assumption of any energy other than the interaction of specific neural elements."

In practice, since Freud believed that the ultimate explanation for behavior was physical, the energic hypothesis came to overshadow the dynamic one. All of his observations of behavior were ultimately "explained" as manifestations of varying quantities of psychic energy. It cannot be emphasized strongly enough that the concept of psychic energy is purely a metaphor. As used in Freudian theory, however, its metaphoric significance has been lost and the construct has taken on all the force of an established fact. In actuality, energic "explanations" are not explanations at all. They are merely tautological restatements of the dynamics in different terms. The ego which blocks awareness of the motivating impulse simultaneously prevents the desired action which would gratify that impulse. To the observer, what is blocked, therefore, is not psychic energy, but an act of behavior; what is expressed in the resultant symptom is a substitute for the action, not the creation of a new channel for the discharge of energy. It is irrelevant to our clinical understanding to posit an energy whose existence can never be demonstrated for behavior which is meaningful only in terms of motivation, psychological mechanism, and ultimate action.

In this part of our paper we have tried to define the criteria for a scientific methodology in psychoanalysis. Scientific usefulness is the only basis for evaluating the assumptions of psychoanalytic theory. Assumptions are useful if they are capable of validation or refutation. In this context, we have examined the early period of Freud's work in which the basic concepts of psychoanalytic theory were established. We have distinguished those concepts which remain a useful part of our thinking from those which, in our opinion, impede the fruitful development of psychoanalysis. In the second part of our paper we shall take up the historical development of the libido theory and the methodological problems created by its underlying assumptions.

REFERENCES

1. Breuer, J. and Freud, S. Studies in hysteria. In *The Standard Edition of the Complete Psychological Works of Sigmund Freud*, Vol. **2.** Hogarth Press, London, 1955.
2. Fechner, G. Elemente der Psychophysik. Summarized by Boring, E., in *A History of Experimental Psychology*. Appleton-Century-Crofts, New York, 1950.
3. Freud, S. Charcot. In *Collected Papers*, Vol. **1,** pp. 11–23. Hogarth Press, London, 1940.
4. Freud, S. The interpretation of dreams. In *The Standard Edition of the Complete Psychological Works of Sigmund Freud*, Vols. **4** & **5.** Hogarth Press, London, 1953.
5. Freud, S. My views on the part played by sexuality in the etiology of the neuroses. In *Collected Papers*, Vol. **1,** p. 279. Hogarth Press, London, 1940.
6. Freud, S. *Three Contributions to the Theory of Sex*. 4th ed. Nervous and Mental Diseases Publishing Co., New York and Washington, 1930.
7. Kardiner, A. *The Individual and His Society*. Columbia Univ. Press, New York, 1939.
8. Kardiner, A. *The Psychological Frontiers of Society*. Columbia Univ. Press, New York, 1945.
9. Lashley, K. S. and Colby, K. M. Comment: An exchange of views on psychic energy and psychoanalysis. Behavioral Sc., **2:** 234, 1957.
10. Levy, D. Fingersucking and accessory movements in early infancy. Am. J. Psychiat., **7:** 881–918, 1928.

HOMOSEXUAL PANIC: CLINICAL AND THEORETICAL CONSIDERATIONS

BURTON S. GLICK, M.D.[1]

INTRODUCTION

It is doubtful if many terms in the lexicon of psychiatry and psychoanalysis have been subject to more variegated usage than that of *homosexual panic*. It is a phrase used often in teaching seminars, case discussions, and in the written descriptions of case histories. One would assume from the frequency of this occurrence that the meaning of the term in all of its connotative and denotative aspects is well understood and well grounded, and that its psychodynamics have been fairly well elucidated by now. It is all the more surprising, therefore, that a perusal of such standard reference works as the Quarterly Cumulative Index Medicus and the Current List of Medical Literature, the former over about the past twenty years, reveals the existence of only one paper (9) devoted specifically to the subject in question. Furthermore, the entity homosexual panic is apparently not recognized as existing by many of the standard psychiatric and psychological dictionaries (10, 12, 15, 18). This paradox is rendered stranger still when one considers how often this entity has been called the culprit terminating (usually suddenly, with the flight of the patient) untold numbers of treatment situations, and precipitating large quantities of psychotic reactions.

This paper is essentially an attempt to clarify this situation; to bring, if possible, some central meaning to a concept that has had many meanings attached to it, ranging from vague feelings of discomfort to truly horrifying fright accompanied by disordered thought and behavior.

It is evident that any consideration of this entity must include both its "homosexual" and "panic" aspects. In the two specific dictionary definitions that the author has been able to find, the first in the Psychiatric Dictionary of Hinsie and Shatzky (16) and the second in "A Psychiatric Glossary" (1), there are already areas of disagreement. According to the former, homosexual panic is "an acute schizophrenic episode, characterized by intense fear, marked excitement, and paranoid ideas; usually of short duration, occurring in individuals whose homosexuality is unconscious." (The implication here is that homosexual panic does not occur in those whose homosexuality is conscious, and that not everyone has contained within him unconscious homosexuality, both of which inferences are not borne out by the facts). According to the latter, homosexual panic is "an acute and severe attack of anxiety based upon unconscious conflicts involving homosexuality." The first definition insists on the presence of a psychosis and stresses also the intensity of the panic aspect, while the second does not consider homosexual panic to be in the nature of a psychotic reaction and is apparently content to describe the panic as an attack of acute anxiety and let it go at that. In one respect are these definitions in agreement: the homosexual conflict must be unconscious.

As for "homosexual" and "panic", neither question is easily disposed of. Let us consider the question of panic first.

THE PANIC

It is incumbent upon us at once to decide whether or not panic is synonymous with acute anxiety, and, if it is not, to determine whatever quantitative and qualitative differences may exist. Certain authors have

[1] Department of Psychiatry, State University of New York, Downstate Medical Center, Brooklyn, New York.

implicitly or explicitly recognized a difference between the two terms. Freud (13) has described panic as a "gigantic and senseless fear" and he goes on to say, "It is not to be expected that the usage of the word 'panic' should be clearly and unambiguously determined. Sometimes it is used to describe any collective fear, sometimes even fear in an individual, *when it exceeds all bounds* (italics added) ... It is impossible to doubt that panic means the disintegration of a group ..." (and by implication, of an individual).

Fenichel (11), who does not directly differentiate between panic and anxiety (calling the former traumatic or primary anxiety), nevertheless refers to "wild panic ... paralyzing panic" in which ego control fails, affect becomes overwhelming, and in which there is a regression to a state of automatic and unspecific anxiety. Brosin (4), in a paper devoted to panic states and their treatment, carefully distinguishes between "strong anxiety and tension" and "full blown terror." Diethelm (8) classifies panics as "extreme cases" of intense fear in which escape or counter-attack as forms of adjustment are impossible for the afflicted individual, and which may lead to the use of many projective mechanisms and the disorganization of the personality with the presence of hallucinations, delusions and a thinking disorder. He states, "Fear and panic, both with a definite content, are fundamentally different from the contentless anxiety."

In a recent article Daly (6) delineates fear as ranging from "intense terror verging on panic to feelings of anxiety or apprehension." It is from his patients perhaps (as it usually is from patients) that we get our most valuable and lucid data. One patient, in describing his fear reaction, speaks as follows. "There was a feeling of impending disaster. It's a horrible sensation; I can't describe it; a feeling that I am going stark, raving mad." Another patient said, "I can't even speak; the terror won't let me." In some of Daly's patients panic evoked a primitive impulse to flee, and the terror of an 18 year old girl was such that she would run to the nearest person and clutch him tightly. When describing less intense emotions his patients would use the words "anxiety" or "apprehension" to characterize their feelings.

Kempf (17), who was apparently the originator of the term *acute homosexual panic*, speaks of "anxiety and even panic." We will discuss Kempf's original concept at length later in this paper.

Clinically then there appear to be good grounds for distinguishing between acute anxiety and panic. At the very least one would have to say that panic is anxiety of such unbearable, overwhelming, all-pervading intensity that it truly does deserve a different name from anxiety. But there is more to the picture than that. There appears to be also a qualitative difference between the two which, although very difficult to describe, must be understood if the full meaning of panic is to be apprehended. In a panic state there is something else besides the well known and often described physiological and psychological concomitants and equivalents of acute anxiety. The terror reaches such massive proportions that the individual is unable to function *at all* (something which is not often true in acute anxiety). He has the impulse to flee, to run madly about in a random, haphazard fashion as if this undirected motor activity would bring some surcease from the agonizing fright; and even to clutch somebody, anybody, in a desperate, sometimes voiceless, sometimes vociferous attempt to preserve his life, identity and innermost integrity. While in this state he is incapable of any logical, objective thought or action. Often the impulse remains but an impulse, as the individual becomes completely paralyzed with fear. At other times the impulse is carried into action.

Questions arise at this point as to whether or not a panic state is itself a psychotic re-

action, and also if such a state can occur in normal individuals. I believe the answer to the first question must be in the affirmative. The entire panic reaction may be considered as a massive regression to an infantile state of disorganized and unspecific activity in which the ego, now rendered quite primitive, is unable to master by any means the powerful stimuli, both internal and external, that affect it (Fenichel's "primary anxiety"). The ego is overwhelmed and out of control and therefore all of its functions, including reality-testing, restraint of instinctual drives, motor control, sensory perception and memory, are likewise seriously disturbed to the extent that life itself is endangered. This is certainly one way, and a classical one, of describing a psychosis. A panic state may be considered then as a psychotic episode, usually brief and almost invariably reversible.

The second question might be phrased thus: Are panic states restricted to those who are psychotic? Fenichel (11) replies in the negative when he says that a traumatic state occurs "in normal persons when they react to danger with paralyzing panic." Brosin (4) adheres to the Meyerian view that panic states are behavior patterns which may occur to almost anyone, and Diethelm (8) asserts that "powerful, threatening conditions" can cause a panic reaction in a well-balanced person.

There really seem to be no valid theoretical or clinical reasons for assuming that panic states can occur only in psychotic or pre-psychotic individuals. All the weight of the evidence is on the other side. Normal persons in groups when exposed to truly catastrophic situations such as battle or fire have panicked; non-psychotic patients in analysis or therapy under the stress of severe unconscious conflicts have panicked; and it is not going very far to affirm that normal individuals, even when not exposed to the contagion of groups, and when not in treatment situations, may panic in perilous real and in perilous psychical circumstances.

THE HOMOSEXUALITY

It is on the question of the homosexuality involved in homosexual panic that significant difficulties and confusion arise. The whole idea of homosexual panic is an interesting one because if one does not accept the postulate of an unconscious mind (and by extension, of unconscious homosexuality), then for him there can be no such thing as homosexual panic.

It is not within the scope of this paper to enter into a polemic on the existence of the unconscious except to say that listening carefully to the productions and associations of patients (especially those who are psychotic) should leave no doubt in anyone's mind concerning its presence, power, and contents. In this connection it is interesting to note that the diagnostic entity "Panic, acute homosexual" is officially recognized in the Diagnostic and Statistical Manual (7) of the American Psychiatric Association and bears the number 082.

In discussing panic reactions, one thinks immediately of acute paranoia because it is evident that if one goes into a panic state it is out of fear that something horrible is happening, or will imminently happen to him, and if there is no real external danger to account for it then the danger must be coming from within. If such is the case and if the patient explains the panic reaction on manifestly inappropriate or distorted premises, then he is obviously unaware of its motivating force which may be said to be truly unconscious, and this apparently holds true in homosexual panic.

To go further into the matter at this point would unduly complicate things. For instance one could speculate that even when the external catastrophic situation is known, and thought to be a fairly appropriate stimulus for a panic response, the cause of the panic is not only the conscious fear of annihilation but also whatever unconscious meanings this particular form of destruction has for the individual (castration, separa-

tion, homosexual assault, loss of identity, etc.). To put it into other words, it may well be that what often produces the panic in a situation of real danger is a combination of conscious mental mechanisms (the real danger) and unconscious ones (the unconscious significance of the real danger) arranged in some form of Freudian 'complementary series' wherein if one factor is present in great amount, less of the other is required to yield the end result.

What is the relationship between such an unwarranted, unrealistically based, autistically determined panic, which is in the nature of an acute paranoid reaction, and unconscious homosexuality? Freud explained paranoia as a defense, via projection, against unacceptable homosexual impulses (14). The concept homosexual panic itself signifies an acceptance of this relationship since it clearly implies that the unconscious homosexual conflict is causing the panic reaction. Although the general applicability of Freud's formulation has been challenged time and again, one can at least say that in *some* cases of paranoia a latent homosexual element does play an important role. In homosexual panic, *by definition*, this must always be the case.

Although clinicians may differ in their appraisal of the relative impact of unconscious homosexuality in paranoia, psychologists have apparently found good evidence for the existence of such a relationship. Aronson (2) observed that, "paranoid subjects report an overwhelmingly greater number of homosexual signs on the Rorschach Test than do either non-paranoid psychotics or normals ... The obtained results appear to be strongly supportive of the Freudian theory of paranoia ... The presence of a large number of homosexual signs on the Rorschach, particularly on a psychotic record, is suggestive of some paranoid involvement." The same held true in his study of the "Blacky" test (3).

Chapman and Reese (5), looking at schizophrenia from the Freudian point of view as a regression from object-libido to ego-libido (narcissism) in which homosexuality or libidinal attachment to others of the same sex is an intermediate step, felt that the schizophrenic Rorschach records they studied showed distinct evidence of homosexuality and markedly elevated scores of homosexual responses as compared to the test protocols of normal control subjects.

We are next faced with the task of determining the nature of the homosexual conflict in homosexual panic and how this conflict expresses itself symptomatically. For this purpose one cannot do better than refer to the work of Kempf.

KEMPF'S CONCEPT

In his book *Psychopathology*, published in 1920 (17), Kempf included a chapter on "The Psychopathology of the Acute Homosexual Panic." In it he ascribes the panic reaction to the "pressure of uncontrollable perverse sexual cravings" and remarks its frequency in situations where men or women are grouped together for prolonged periods as in army camps, prisons, schools and asylums.

Kempf's explanation of the more superficial aspects underlying this disorder is at times obscure. He believes truly enough that "the perverse sexual craving threatens to overcome the ego, the individual's self-control," but then accounts for this by saying that "the affections for winning social esteem have been pushed into eccentric adjustment." It is not evident to me what this statement signifies but I suspect that what Kempf means here is an adumbration of what he later beautifully puts as, "The patient is telling the physician [by means of his symptoms] that he has lost control of his sexual cravings which are forcing him to offer himself as a sexual object."

Kempf makes much of feelings of inferiority and weakness of the ego through fatigue, debilitating fevers, loss of love-object, misfortunes, etc. as causative factors, tending to neglect the more funda-

mental issue of chronic ego-enfeeblement due to constitutional or developmental factors and conversely of the undue strength, again possibly constitutional, possibly developmental, of the latent homosexual drives. It is doubtful also if this condition occurs only in those with pathologically pronounced conscious or unconscious inferiority feelings, although this would certainly be a factor in many cases since these feelings might lead one to wish to submit.

The crux of the matter is stated by Kempf as follows. "The cause of the anxiety and panic is the uncontrollable, perverted... craving struggling with socialized cravings, the ego, in the same personality. When the sexual cravings can not be controlled they become disowned by the ego as a foreign influence, and the ideas and visions, or sensations they cause, are treated as being due to a foreign influence."

That is to say projections take place which are exactly in the nature of all psychotic paranoid projective mechanisms, and as a result, "The perverse affective craving causes delusions about, and hallucinations of, situations, objects, and people which tend to gratify the craving. Horrified he [the patient] is swept off his feet into a hell of hallucinated temptations and demons of destruction."

In discussing these matters Kempf unfortunately gets involved in his own theoretical speculations which lead him to posit that an "affective disturbance" may be due to "repressed autonomic tensions" (rather than repressed psychological or psychobiological drives) and that during sleep indigestible food causes "increased gastro-intestinal striving" (rather than increased gastro-intestinal sensations triggering off, or being used by, unconscious wishes to form a dream or symptom). However, he is correct in saying, "When the erotic hallucination is felt to be an external reality, and no defense is found, panic ensues."

Now, how do the symptoms, which are the projected realizations of the patient's unconscious need to offer himself sexually, express themselves in homosexual panic? Kempf presents a series of 17 illustrative cases culled from a total of several hundred from which we can formulate the principal symptomatology. These patients ranged in age from 18 to 43 years with a mean of 27.5 years. There were 15 males and two females. All the males with one exception were members of the United States armed forces.

The cardinal symptom appears to be delusions of influence and control, of being used in various ways, in short of being unwillingly under the forceful control of certain others for various dire purposes. This takes diverse forms. Thus patients would complain of being "doped," "framed," "hypnotized," injected with drugs, tortured, poisoned, shot through with electricity, threatened with violence, death or sexual assault, and of having their manhood removed or their genitals altered.

In some cases the homosexual element is directly expressed. One patient complained that he had been subjected to a sexual assault and that his penis was "all shriveled up." Another had the delusion that "white stuff" which he characterized as "come" had been put into his food. A third felt his "manhood" had been taken from him and he could not sleep for fear of an impending sexual assault. Still another felt that sodomy had been performed on him which destroyed his sexual powers.

Sometimes the homosexual component is expressed symbolically. The complaint about poisoned food may often be a disguised representation of an unconscious fellatio phantasy. One patient put a white paste into his mouth under someone's "hypnotic" influence; another felt there was a "sticky substance" in his mouth. In both these cases the orally incorporated substance may well be seminal equivalents. Anal material, the result of unconscious wishes to be anally penetrated may readily be presented directly or symbolically. For instance, patients felt that sodomy had been

performed on them, that they had received injections, or that they had "gonorrhea of the rectum." One patient remarked (in reference to those he felt were controlling him), "They said the Major was giving me salt enemas and eggnogs." One would have to go far to find a clearer picture, symbolically presented, of a combined fellatio and anal-penetration phantasy. Another patient had the idea that a sergeant intended to blow up the stables where the patient slept. Still another complained that "they" pumped him full of air and then, "They would learn me how to bend over and it would go away."

At times there appears to be neither a direct nor a symbolic representation of any homosexual phantasy, but instead an apparently asexual picture of undue malignant influence, physical violence or impending death. Thus a patient might complain that he was being "doped", tortured or crucified, or that certain persons were plotting his death. An objection might be raised at this point to considering this type of material, if it alone occurs without an intimation of anything sexual, as fitting criteria for a diagnosis of homosexual panic. In this connection I would like to introduce Kempf's remark that he had never seen an aggressive male or female homosexual in a panic. The idea inherent in this statement is one of the most important for an understanding of the disorder. We cannot be quite certain of what Kempf means by "an aggressive homosexual." He might mean merely an *overt* homosexual, using the words aggressive and overt synonymously, or he might mean an overt homosexual who takes the man's role exclusively, here using maleness and aggressiveness synonymously. In either case I do not think the statement can stand as being universally valid. Many of us have seen overt male homosexuals of all types, the masculine (active), feminine (passive), and mixed, develop acute paranoid reactions, and this raises the interesting problem of how an overt male homosexual, let us say, to make the dilemma more striking, of the feminine type, could enter into an acute homosexual panic.

In order to understand this we must realize that manifest (conscious) and latent (unconscious) homosexuality are not one and the same thing[2]; that they exist, speaking metaphorically and topographically, in different "areas" of the psychical system; that they pursue independent courses (the contents of the unconscious may always be considered to be unavailable to the ego and conscious mentation except through interpretation, and sometimes never available to consciousness under any circumstance), and that in a sense "never the twain shall meet." It is probably in error therefore to speak of a patient's unconscious homosexuality or homosexual wishes becoming conscious. When an overt homosexual develops a paranoid reaction it is on the basis of his *unconscious* homosexuality (which might best be described as a passive-feminine attitude or identification) provided there are present in this reaction homosexual elements. The unconscious, infantile wish in all cases of homosexual panic in males is to submit, or offer oneself, sexually to the father, and this wish, for whatever its realization means to the patient (castration, turning into a woman, complete subjugation with loss of will, physical pain) is energetically repudiated by the ego and through the mechanisms of reversal of affect (love into hate) and projection is made to appear as some horrifying threat coming from the outside. It is a matter for speculation and something of a mystery why in an overt, passive-feminine male homosexual such an unconscious, infantile wish should provoke terror, while at the same time a form of behavior which is *apparently* the fulfillment of this wish is acceptable to the ego and even desired. I would guess that it is in the unconscious

[2] The core idea of the sharp dichotomy between manifest and latent homosexuality, and its applicability to paranoid states, was originally presented to the author by Drs. Irving Rubins and L. Peter Deutsch.

associations to the wish (the aforementioned castration, subjugation, etc.) that the terror provoking mechanism lies, and that conscious homosexual behavior is free of these elements, which remain attached to the original unconscious wish until they break through into consciousness in the psychotic episode. The original desire to sexually submit apparently always remains unconscious.

In women, if one is to judge from the following productions of one of Kempf's female patients, the persecutor is the woman with the phallus, originally the phallic mother. This patient misinterpreted a physical examination administered to her by a woman physician as a sexual assault. "She did something to me... she stuck me in the thigh. She said I want to show you a little invention of my own [the phallus]. She put a towel over my face and asked me what she had in her hand.... You destroyed something you can't replace [virginity]. You can't try that holy mother [the phallic mother] business on me.... She drove something into me with a tack hammer. I never wanted a girl [a reference to homosexual relations]."

It is possible that in the male too, standing behind the father, and at even deeper unconscious levels, is the phallic mother, who then would be the true, original persecutor in all cases of homosexual panic, and the one from whom the sexual gratification is craved.

In returning now to our original objection against considering non-sexual content sufficient for a diagnosis of homosexual panic, we must bear in mind that in all instances where material of this kind is presented, the patient is always the passive recipient. Horrible things are being done *to* him, against his will, and he is powerless to defend himself against these controlling forces. In this sense then the patient is exhibiting the passive-feminine side of his nature which may be considered a derivative of the original unconscious passive-feminine strivings toward the father. This however may be an unjustifiable assumption and one not in accordance with the facts.

Admittedly it is a difficult thing to make a diagnosis of acute homosexual panic when there is no presentation of any homosexual material either directly, obliquely or symbolically, and in such cases one rightfully should be reluctant to make this diagnosis, which fails to take into account the other great instinctual drive (besides the sexual); that of aggression. In a healthy personality the sexual and aggressive drives are fused and balanced. In a psychotic personality there is either a split or an imbalance, and there is no particular reason why the aggressive drives should not predominate as often as the other. We should have the right to speak of an *acute aggression panic*. From this discussion it is obvious that one of the chief difficulties in making the diagnosis of acute homosexual panic is that it is as much an interpretation as it is a diagnosis.

The other principal symptoms of this entity, in addition to the delusions of influence and control, are: auditory hallucinations, frequently with a direct sexual content of a derogatory, castrative or accusative nature; visual hallucinations (one patient saw snakes biting him on the chest); ideas of reference usually with a sexually perverse or homosexual content; delusions of grandeur; olfactory hallucinations; and a thought disorder. It goes without saying that a true panic state as previously described, and not merely anxiety, tension or depression, must be present. In some of Kempf's case descriptions apparently this was not always true. However, it is possible that he simply neglected to emphasize or mention the evidences of panic in these cases. Where he does mention them, they are clear enough. One patient cried, shouted and cursed; threw his arms around an attendant and begged for protection. Another jumped from his bed, fell on his knees and begged to be saved by the physician. A third pounded on the door and called for help.

Catatonic paralyses brought on by intense fear were also seen.

Delusions, hallucinations, ideas of reference, thinking disorders, bizarre ideation; these are all symptoms of a schizophrenic process which accurately describes what a homosexual panic is. It can be seen from a description of the symptoms that homosexual panic differs in no way from any other acute schizophrenic state except perhaps content-wise in the predominance of sexual material.

Concerning the prognosis Kempf says the following. "The acute dissociation of the personality, in both sexes, may become chronic and run a protracted course, varying from several weeks to many years, with final recovery, or may become permanent without further deterioration; or pursue a course of progressive deterioration." The same may be said for all cases of acute schizophrenic reaction and so is not peculiar to homosexual panic. He maintains further that the prognosis of a homosexual panic is usually favorable for that episode, but the future of the patient is "most insecure" unless he obtains "insight and a fortunate sexual adjustment". Indeed wherever Kempf's account of his 17 cases includes an estimation of the prognosis it is likely to be unfavorable. One comes frequently across such remarks as, "The prognosis is apparently poor just in so far as his homosexual cravings tend to break through his concerted efforts to control them." Where one patient asserted that he would rather die or go crazy than become a fellator Kempf added that one or the other of these preferred alternatives would be the likely outcome.

We see then that the acute homosexual panic, like any other acute schizophrenic reaction, may be the first manifest phase of a beginning schizophrenic illness, may represent a stage in an already established schizophrenic condition, or may exist as an isolated instance in the course of an otherwise normal life. Kempf does not believe that homosexual panic states are restricted to those who are psychotic. "Irrepressible homosexual cravings are not characteristic of the ignorant and mentally defective. Under appropriate sentimental conditions, homosexual cravings probably may become aroused in most males and cause very serious disturbance of self-control, developing at times into nothing less than a psychosis."

CONCLUSION

We are now in a position to attempt a concise and relatively complete definition of this entity. *Acute homosexual panic is an acute schizophrenic reaction, usually temporary in duration, displaying the full panoply of schizophrenic symptoms accompanied by sensations of intense terror manifesting themselves in wild excitement or catatonic paralysis. It is based upon the patient's fear of loss of control of unconscious wishes to offer himself as a homosexual object which he feels will result in the most dire consequences.*

It will be seen that this definition, with the added clarification of the homosexual element, agrees closely with that found in the Psychiatric Dictionary of Hinsie and Shatzky (16).

SUMMARY

Homosexual panic is a subject which has been little touched upon in the literature despite its frequent mention in case discussions. Any consideration of this entity must include both its "homosexual" and "panic" aspects.

Panic should be clearly differentiated from acute anxiety both quantitatively and qualitatively. A panic state is a psychotic reaction which can occur in normal individuals. An unrealistically based, autistically determined panic is in the nature of an acute paranoid reaction, which, according to Freudian doctrine is based upon unconscious homosexual impulses.

Kempf (17) introduced the concept of acute homosexual panic in 1920 and believed it was due to the "pressure of uncontrollable

perverse sexual cravings" which were forcing the patient to offer himself as a sexual object, and which the patient disowned via the mechanism of projection with all of its attendant psychotic symptomatology.

Acute homosexual panic is in reality a type of acute schizophrenic reaction which is heavily laden with homosexual material presented either directly, obliquely or symbolically. Where no homosexual content is to be found, but instead a picture of undue malignant influence, physical violence or impending death, the use of the term *acute aggression panic* is suggested.

It is entirely possible for an overt male homosexual of the passive-feminine type to develop a homosexual panic because the latter evolves from *unconscious* homosexuality which exists on a different psychical plane from that of conscious homosexuality, and which has associated with it many frightening unconscious phantasies.

One of the difficulties in making the diagnosis of acute homosexual panic is that it is as much an interpretation as it is a diagnosis.

The prognosis for this disorder is the same as for any other acute schizophrenic state, and under appropriate conditions it may occur in the course of an otherwise normal life.

An attempt is made to offer a concise definition of this entity.

REFERENCES

1. *A Psychiatric Glossary*. Committee on Public Information. Am. Psychiat. Assoc., Washington, 1957.
2. Aronson, M. L. A study of the Freudian theory of paranoia by means of the Rorschach Test. J. Project. Tech., **47**: 397–411, 1952.
3. Aronson, M. L. A study of the Freudian theory of paranoia by means of the Blacky Pictures. J. Project. Tech., **17**: 3–19, 1953.
4. Brosin, H. W. Panic states and their treatment. Am. J. Psychiat., **100**: 54–61, 1943.
5. Chapman, A. H. and Reese, D. G. Homosexual signs in the Rorschach. J. Clin. Psychol., **9**: 30–32, 1953.
6. Daly, D. Ictal affect. Am. J. Psychiat., **115**: 97–107, 1958.
7. *Diagnostic and Statistical Manual, Mental Disorders*. Am. Psychiat. Assoc., Washington, 1952.
8. Diethelm, O. The nosological position of panic reactions. Am. J. Psychiat., **13**: 1295–1316, 1934.
9. Dinerstein, R. H. and Glueck, B. C. Subcoma insulin therapy in the treatment of homosexual panic states. J. Soc. Therapy, **1**: 182–186, 1955.
10. English, H. B. and English, A. C. *A Comprehensive Dictionary of Psychological and Psychoanalytical Terms*. Longmans, Green, & Co., New York, 1958.
11. Fenichel, O. *The Psychoanalytic Theory of Neurosis*. Norton & Co., Inc., New York, 1945.
12. Fodor, N. and Gaynor, F. *Freud, Dictionary of Psychoanalysis*. Philosophical Library, New York, 1950.
13. Freud, S. *Group Psychology and the Analysis of the Ego*. Standard Edition, Vol. XVIII. Hogarth Press, London, 1955.
14. Freud, S. *Psychoanalytic Notes upon an Autobiographical Account of a Case of Paranoia*. Collected Papers, Vol. III. Hogarth Press, London, 1953.
15. Harriman, P. L. *The New Dictionary of Psychiatry*. Philosophical Library, New York, 1947.
16. Hinsie, L. E. and Shatzky, J. *Psychiatric Dictionary*, Part 2 (Supplement). Oxford University Press, New York, 1953.
17. Kempf, E. J. *Psychopathology*. C. V. Mosby Co., St. Louis, 1920.
18. Podolsky, E. *Encyclopedia of Aberrations*. Philosophical Library, New York, 1953.

THE CONCEPT OF NEMESIS IN PSYCHONEUROSIS

A. H. CHAPMAN, M.D.[1]

INTRODUCTION

The central core of some psychoneurotic disorders is the patient's feeling that he is destined to repeat in his life the pattern of some other person's life, usually to a tragic end. Thus the patient may feel that he is destined to die or to become insane at some time, following the life pattern of his mother, father, or some other person emotionally significant to him. This feeling, or conviction, goes far beyond the "talion principle" in which the patient feels that past misdeeds and hostile thoughts must of necessity draw down upon him the retributive justice of a tit-for-tat punishment similar to any misfortune that befell the person toward whom he had hostile feelings. The feeling of being destined to follow the course of another's life to a similar catastrophic end embraces the conviction that there is an extensive life pattern similarity, even in correlations of events and ages when they occur, over periods of many years or perhaps the entire lifetime from adolescence onward. So far as I am aware, this type of emotional problem has not been specifically dealt with previously in psychiatric literature, and it has no particular name or designation. Yet, it is found from time to time as one explores the emotional structure of various types of neuroses.

For purposes of discussion, it will be convenient to give this type of problem a concise name. It will be referred to as the nemesis feeling, or conviction, after the Greek goddess Nemesis, whose task it was to pursue wrong-doers relentlessly and inflict upon them punishments often similar in nature to their misdeeds. The term nemesis was also later used in Greek ideology to designate a process of retributive justice inherent in the nature of things, applying not only to individuals but also to city-states and nations. The term nemesis feeling is therefore a concise, yet accurate description of the emotional problem we are discussing.

[1] Department of Psychiatry, University of Kansas School of Medicine, Lawrence-Kansas City, Kansas.

SIGNIFICANCE OF NEMESIS FEELING

The nemesis feeling has marked significance and force for the person disturbed by it. He feels that he is being carried forward through life on a conveyor belt at a speed over which he has no control. The things that are happening to him as he is carried along have all been previously determined by the events of the life of the person whose pattern he is following and he is helpless to struggle. At the end is a painful catastrophe, such as untimely death, insanity, or a dreaded disease.

The person whose life pattern is being followed is usually dead, though occasionally alive but removed from society by long term institutionalization in a mental hospital. This person is almost always the father or mother, and usually, but not always, the parent of the same sex as the patient. A woman disturbed by the nemesis feeling, for example, may fear that she is following her mother's pattern and a man may fear that he is destined to parallel his father's course.

There are always certain similarities of at least a superficial nature in the life patterns of two members of the same family. These similarities seem to the patient to confirm the reality of his nemesis fears. At other times the patient seems unconsciously to act out certain details to make the life patterns more similar. Discordant features of the two involved life patterns do not dissuade the patient from his fear, but rather are overlooked or discounted.

I have had the opportunity of studying intensively and over long periods of time, five patients in whom the nemesis feeling was a central feature of their neuroses. These patients were seen in psychotherapy. More superficial studies, limited to eight to ten interviews, were made on two other patients who showed this feature but who declined to enter into more extensive psychotherapeutic exploration of their problems.

Fragmentary nemesis feelings are seen commonly in psychoneurotic patients who feel that in some relatively restricted area of their lives the nemesis principle holds, but that the other areas of their lives are free of such influence. I have observed such fragmentary nemesis feelings in nine patients. They are not being specifically considered here, but only those patients in whom the nemesis feeling was a pervasive factor spreading into most areas of the patient's life. However, what is true of the nemesis feeling in the more pervasively affected patients is true in a more restricted sense in those patients affected in one area of activity only; the restricted nemesis feeling is smaller and more localized, but otherwise similar. It may, for example, involve only the marital or vocational area, but not affect the other aspects of the patient's existence.

The nemesis feeling was usually, but not always, at least partly conscious in these patients, though they became aware of its far-reaching ramifications only in psychotherapy. None had ever previously told another person of this feeling, and so far as I am aware none of these patients subsequently confided their nemesis feelings to another person. They felt that their nemesis feelings, though realistic and reasonable to them, would seem extremely bizarre to others.

The presenting symptomatology of these patients was varied. Phobic, anxiety and psychosomatic symptoms predominated, but depressive and obsessive problems also occurred. The nemesis feeling came to the fore as a rule only as psychodynamic exploration proceeded deeply into the patient's inner world of feelings, anxieties and fantasies.

Rorschach studies on these patients revealed no signs of schizophrenic tendencies and none of them showed clinical signs of schizophrenia. Depressive trends were present in all the patients in varying degrees, but were a major problem in only one patient. This patient became suicidal and had to be hospitalized for eight weeks. With this exception none of the patients became psychotic or showed psychotic tendencies at any point.

All the patients with this problem were between the ages of 20 and 35. Both sexes were represented in this group, and all the patients were married and had at least one child. Though they may have been aware of their nemesis fears before, there seemed to be a tendency for such fears to become more acute after the patients married and had families of their own. The stage seemed to them to be then set for the final enactment of the catastrophic end of the nemesis sequence.

On the whole, these patients were interesting but difficult persons with whom to work. Psychotherapeutic progress was slow. Though each of them was appreciably helped, none of them completely lost his symptoms. The nemesis fear was much ameliorated in each patient, but vestiges of it still remained in them regardless of the length of therapy: two of the patients were seen for more than four years, but the others were seen for shorter periods.

An extensive exploration of the nemesis feeling revealed its roots to lie always in a profound feeling by the patient that he had been directly and solely responsible for the death or insanity of the person whose life he was doomed to imitate. Powerful feelings of guilt formed the emotional forces which propagated the nemesis feeling and gave to it its long tenacious hold over the patient. Such feelings of guilt had, of course, no validity in the actual causes of death or psy-

chosis in the person involved; they had validity only in the fantasies of the patient. The nemesis feeling had its origin usually in the death or psychosis of a parent during the childhood of the patient. Hostile or competitive feelings toward the involved parent were present in half the patients studied.

CASE REPORTS

More detailed aspects of the nemesis feeling will be illustrated by presenting briefly two representative case studies.

Case 1. When first seen, this thirty year old married woman had for the previous year been suffering from severe anxiety attacks, depressiveness, and obsessive ruminations concerning cancer, insanity, suicide and a fear of harming other persons. She had received a variety of medications and supportive medical care, but none of this had helped her.

The life history of the patient, as unfolded in intensive psychotherapy during the next two years, revealed that the patient's mother and father, a physician, had been divorced when the patient was three years old. When the patient was seven, her mother remarried. When she was twelve, her step-father began to make sexual advances to her and over the next several years, frequent seduction and actual intercourse occurred.

When the patient was fourteen, her mother became aware of the sexual activity that the step-father was having with her daughter. The nature and depth of disturbance that this caused in the mother is not clear, but during the next six months she developed a schizophrenic psychosis and was hospitalized in a state psychiatric hospital. The patient continued to live with her step-father for another year and a half after this, but at the age of sixteen she was sent to a distant city to live with her true father and his second wife.

The patient's mother never recovered. During the patient's late teens and early twenties, she attempted several times to take her mother home on trial visits from the hospital, but such attempts were unsuccessful. The mother's psychotic behavior on each occasion made re-hospitalization mandatory. Since the patient was twenty-four her mother has been continually in a state psychiatric hospital.

The patient, in her early twenties, received secretarial training and worked in a medical center where she met and married a young physician. Within two years after marriage, she gave birth to her first child, a girl, and fourteen months later, to a boy. The onset of her symptoms apparently came during the last trimester of her second pregnancy.

The nemesis fear of this patient, that she was destined to lead a life pattern similar to her mother's, terminating in incurable insanity and institutionalization in a mental hospital, had been present with the patient since the onset of her symptoms. It was a conscious fear and it underlay all her other symptoms. She felt that, like her mother, she had married a physician and was proceeding to have the children who would some day cause in her the same psychotic development she felt she had caused in her mother. All the details of the sexual affair with her step-father had been repressed and he had been idealized as a wise and kindly man. The memories of his actual role were recovered in psychotherapy.

In psychotherapy, extending over a period of two years, the details of the life history and associated feelings were explored. The patient made a great deal of symptomatic progress. Her therapy was terminated when she moved to another city. Though much improved at that time she occasionally had mild recurrences of her anxiety and her nemesis fears.

Case 2. The patient was a forty year old, married businessman who was referred for psychiatric consultation because of severe anxiety attacks during which he feared death, suffocation, and impending insanity. These symptoms had been present for three

months and supportive medical care by his family doctor had not been helpful.

The patient had been the youngest of three boys whose mother had died when the patient was eleven years old. His father had remarried several years later. After public school education the patient held a variety of jobs before he went into business for himself in his late twenties. He had been successful in business and was active in civic and political affairs in his community. He had married in his middle twenties and had three daughters ranging in age from nine to fourteen.

The patient's symptomatology underwent a good deal of improvement during psychotherapy which was limited to a discussion of current situations in his life; a major part of treatment dealt with helping the patient become more expressive of angry and aggressive feelings. He had been previously unable to verbalize anger and had kept assertive feelings bottled up within himself. On this relatively superficial level of treatment, the patient's symptoms improved from their previously incapacitating dimensions to the point where he could carry on his business and family activities with relative comfort. However, he had periodic mild recurrences of his symptomatology in which he again feared death, insanity and suffocation.

Treatment had been restricted to the superficial level described above because the patient had a complete amnesia for details concerning his mother. Though his mother had died when he was eleven, he could remember nothing of her, except a tableau memory of her lying quietly in bed looking at the ceiling.

The patient was anxious to make further progress with his difficulties, but the pervasive nature of his amnesia led to a virtual stalemate in treatment which could not be broken by any kind of associative or anamnestic technique. Because of this difficulty I suggested the extraordinary expedient of asking the patient to get certain outlines of factual data about his mother from other relatives.

The data which the patient obtained from his older brothers was very revealing and indicated a nemesis problem which had clearly been present during the patient's chiidhood and which had apparently been unconsciously operative in his present illness.

The patient had been an unplanned child. His mother had become quite ill at the time of her pregnancy and delivery of him and had never recovered her health. The patient was blamed by his mother and her relatives as the cause of her life-long invalidism after his birth. The mother's symptoms were largely pulmonary, consisting of shortness of breath and profound fatigue.

When the patient was three years old, his mother became psychotic and was hospitalized in a nearby sanitarium after a period of floridly psychotic behavior at home. During her illness the patient was taken to see his mother in the hospital. The experience greatly frightened him since his mother was disturbed and irrational at the time. During the next year or so the mother gradually recovered from her psychotic episode but remained a semi-invalid for the rest of her life. All of the activities of the family were centered around her care. The precise nature of the mother's illness was never clear, but it is probable that most of her difficulty was on a psychiatric basis. When the patient was eleven, his mother contracted lobar pneumonia and died at home after an illness of several days.

After this information had been obtained from his older brothers, the patient recovered the memory that, following his mother's death he had the nemesis feeling that, inasmuch as he allegedly had been responsible for her invalidism, pulmonary disease, insanity, and susceptibility to death from pneumonia due to her weakened condition, he could only pay back this debt by follow-

ing a similar life course. For a period of a year or two in puberty and early adolescence the patient had this painful obsession, which then became unconscious, and remained so until it was recovered in psychotherapy.

This material explained why the patient had feared for himself death, insanity, and suffocating pulmonary disease, for these recapitulated his mother's illnesses and the final fatal outcome. Careful correlation, moreover, revealed that the age at which the patient had the onset of his symptoms was the precise age at which his mother had the onset of her psychotic episode. It became apparent that the patient feared that he would precisely follow the pattern of his mother's life and develop similar illnesses at the exact ages that his mother had suffered her sicknesses.

Upon elucidation of this material, the patient had a great deal of improvement. Subsequently he has been seen in occasional follow-up visits and has remained largely free of symptoms. There have been mild recurrences of his symptoms at infrequent intervals, however, and he has never become entirely free of his symptomatology.

DISCUSSION

These cases demonstrate various features of the nemesis feeling. In the first case, the patient's nemesis fears were conscious for an extensive period of time and were centered around the parent of the same sex. In the second patient the nemesis fears were unconscious during the greater part of the patient's life but were clearly operative in the subsequent emotional illness of the patient. Both of these patients were greatly improved, but not entirely rid of symptoms, by elucidation of the nemesis core in their neuroses. The exploration of the nemesis feeling was the crucial part of the treatment of each patient and without exploration of the nemesis problem it is doubtful that lasting and extensive improvement would have occurred in them. In each patient the exploration of the nemesis feeling was extremely difficult. In the first patient it came only after careful and prolonged analysis of dreams and was pieced out item by item over a period of many months. In the second patient it was brought to the surface only by the unusual expedient of seeking information from relatives.

Nemesis feelings may be easily overlooked in psychotherapy, for the patient has great difficulty in verbalizing them even when he is aware of them. In general, the patient fears that verbalizing the nemesis concept will tend to hasten its final enactment. It is as if to pronounce the words defining the nemesis fear would bring down its implied consequences. When I verbalized the nemesis fear to one patient she anxiously acknowledged the existence of such fears and indicated she had known it for many years but hastened to add, "You are the one who said it, doctor," as if to separate herself quickly from the consequences of having enunciated such a concept.

So fearful are these patients of verbalizing this fear that the therapist often begins to suspect it only when the patient in his dreams repeatedly identifies himself clearly with a deceased person. Usually the final piecing together and interpretation of the nemesis fear is made by the therapist, and the patient quickly and specifically acknowledges the validity of the nemesis fear with a mixture of relief and anxiety. Then the far reaching ramifications of it may be gradually worked out.

The talion fear is a much more limited and vague feeling of a basically similar nature (1). It is the nemesis feeling limited to a single act and its feared "eye for an eye" retribution. It does not embrace a more general life pattern scope as the nemesis feeling does. It is probable that some talion fears are actually fragments of a larger nemesis fear in which only the isolated talion fear was verbalized and the larger neme-

sis feeling of which it was a part remained unexplored.

The nemesis feeling, like many psychoneurotic symptoms (2, 3), is an unsuccessful attempt to solve an emotional problem. It is an attempt of the patient to solve the problem of his relationship to a significant person in his life. Toward this person now dead or otherwise removed from his active life, the patient feels a profound guilt which he has not been able to resolve. He resolves it in a symptomatic fashion by feeling that an imperative destiny requires that his life be similar to the other person's life along with whatever pain and misfortune such a course may bring. In this way the score is settled and the unfinished interpersonal business ended. Out of, and alongside, such a central neurotic problem phobic, anxiety, psychosomatic, or obsessive symptoms may arise, but there is little opportunity for these symptoms to be ameliorated and removed until the central nemesis fear has been elucidated and worked through.

SUMMARY

The term nemesis feeling is introduced to designate a feeling or conviction by a patient that he is destined to follow in his life pattern the general outlines of some other person's life, usually to a tragic end. It is rooted in the profound guilt feelings the patient has over what he feels his role was in the precipitation of the insanity or death of a parent during the patient's childhood. Though phobic, anxiety, psychosomatic or obsessive symptoms may be the overt difficulty, the central problem remains the nemesis feeling which must be explored and worked through to affect the basic pathologic process.

REFERENCES

1. Fenichel, O. *The Psychoanalytic Theory of Neurosis*. W. W. Norton & Co., New York, 1945.
2. Freud, A. *The Ego and the Mechanisms of Defense*. Hogarth Press, London, 1936.
3. Sullivan, H. S. The meaning of anxiety in psychiatry and in life. Psychiatry, **11**: 1–13, 1948.

RUPTURED ANEURYSMS OF THE SUPRACLINOID PORTION OF THE INTERNAL CAROTID AND OF THE MIDDLE CEREBRAL ARTERIES[1]

ASAO HIRANO, M.D.,[2] KEVIN D. BARRON, M.D. AND
H. M. ZIMMERMAN, M.D.

According to Walton's extensive review (11), 30 per cent of intracranial aneurysms arise from the terminal portion of the internal carotid artery, including its major bifurcation, while 28 per cent take origin from the middle cerebral artery within the Sylvian fissure. These percentages were derived from a series of 1,932 ruptured and unruptured intracranial aneurysms. Walton's personal series comprised 145 cases. The remaining 1,787 aneurysms included in his statistics were culled from the numerous reports in the literature.

Despite the evident frequency with which internal carotid and middle cerebral artery aneurysms are encountered, scant attention has been paid to the pathologic effects produced by their rupture. Within recent years 17 examples of ruptured aneurysms in the locations described have been examined at autopsy in Montefiore Hospital. Analysis of this material has revealed three distinct pathologic groups. The first was characterized by massive bleeding into the subarachnoid space, without penetration of brain tissue. In the second group, hemorrhage resultant from aneurysmal rupture lead to disruption of cerebral tissue and bleeding into the ventricular system. With one exception (Case 10), ventricular hemorrhage followed upon destruction of the uncus and fimbria hippocampi with penetration of the bleeding into the temporal horn of the lateral ventricle. The third group was characterized by bleeding into the Sylvian fissure and formation of a hematoma, more or less localized within the leptomeninges. Intracerebral extension of the hemorrhage occurred in this third type of case by disruption of the cortex of the island of Reil and pars opercularis, but the hemorrhagic penetration was usually limited to a short distance. The differences in pathologic anatomy exemplified by these three groups of cases were reflected in clinical and radiological distinctions which may be of assistance to the physician in diagnosis and therapy.

[1] The Henry and Lucy Moses Research Laboratories of the Laboratory Division, Montefiore Hospital, New York, N. Y. The authors gratefully acknowledge permission to use Case 8 to Dr. James Berkman of the Long Island Jewish Hospital, N. Y. Mr. Antol Herskovitz prepared the photographs.

[2] Trainee of the National Institute of Neurological Diseases and Blindness of the National Institutes of Health, Public Health Service, Bethesda, Maryland.

CASE REPORTS

GROUP 1

Case 1. (M. H. 66897) This 52 year old woman had had occasional episodes (1–3 per month) of brief loss of consciousness for two years. These episodes were unassociated with convulsions or automatic phenomena. There was no other noteworthy symptomatology. Five days prior to admission, she complained of severe headache and vomiting. These symptoms cleared spontaneously. On the day of admission, she suddenly vomited and lost consciousness. Hospitalization was immediately effected. On examination, nuchal rigidity, early papilledema, minimal right central facial weakness and a mild paresis of the right extremities were found. The blood pressure was 170 mm. Hg systolic and 80 mm. diastolic. A lumbar puncture was performed. The initial pressure was 330 mm.

of H_2O and the cerebrospinal fluid was grossly bloody. The patient was stuporous for three days. Coma then supervened and was accompanied by bilateral Babinski signs. Because of the worsening of the patient's condition, a left carotid arteriogram was done. This procedure disclosed an aneurysm of the left internal carotid artery just proximal to its major bifurcation. The anterior and middle cerebral arteries were normally disposed. Ligation of the left common carotid artery was carried out in the neck, and immediately following ligature a left subtemporal decompression was performed. During the latter operation the cerebrum was noted to be under increased pressure, and blood was evident in the subarachnoid space. A needle was passed into the left ventricle and clear colorless cerebrospinal fluid was obtained. The patient expired 15 hours after surgery and six days after admission.

Postmortem examination: The general organs did not show significant abnormality. Cerebral swelling was indicated by diffuse flattening of the gyri and by herniation of the unci and cerebellar tonsils. The cerebral cortex underlying the surgical defect exhibited evidence of herniation. The circle of Willis showed the usual configuration and the arteries were thin-walled and marked by only occasional atheromata. There was massive subarchnoid hemorrhage at the base of the brain which spread over the convexities. A 1 cm. aneurysmal sac, which had a thin wall and contained freshly clotted blood, arose from the main trunk of the left internal carotid artery below the bifurcation. It projected medially and presented a large defect in the ventromedial aspect of its wall. An extensive zone of lateral frontal cortex in the neighborhood of the left insula was the site of hemorrhagic infarction. There were no associated arterial occlusions and no evidence of direct intracerebral extension of the subarachnoid bleeding. The ventricular system was normally disposed and did not contain blood. The pontine tegmentum was the site of small, recent hemorrhages.

Case 2. (M. H. 93887) This 55 year old woman was in excellent health until two years before admission, when she had her first episode of subarachnoid hemorrhage and was comatose for five days. Carotid arteriography was negative at that time. The patient recovered completely and remained well until 42 days prior to her death when she suddenly lost consciousness while eating and was hospitalized elsewhere. Examination disclosed nuchal rigidity and coma. The blood pressure was 170 mm. Hg systolic and 80 mm. diastolic. Grossly bloody spinal fluid was obtained by lumbar puncture. The patient regained consciousness five hours later. During the following 34 days, there were three more episodes of subarachnoid hemorrhage, the fifth occurring eight days prior to death. Immediately after the third episode of hemorrhage the patient was admitted to Montefiore Hospital where examination revealed papilledema and a hemorrhage in the vicinity of the right optic disc. There were no lateralizing signs. Carotid and vertebral angiography was done. A small aneurysm was demonstrated by oblique views at the junction of the right internal carotid artery and the posterior communicating artery. Six days prior to death, a sixth attack of subarachnoid hemorrhage developed. She recovered gradually. The following day a Selverstone clamp was applied to the right common carotid artery and was completely occluded. Five hours after this procedure, the patient became totally unresponsive and remained comatose until her death.

Post mortem examination: The findings were similar to those of Case 1, except that cerebral infarction was not present. A 5 mm., oval aneurysm arose from the medial wall of the right internal carotid artery near the junction of the posterior communicating artery. The aneurysm contained a small atheromatous plaque and its lateral

wall showed a point of rupture. The right optic nerve and the aneurysm were adherent. Again, there was subarachnoid hemorrhage without penetration of the brain. The pontine tegmentum presented several hemorrhages. The lumen of the right common carotid artery did not contain a thrombus.

Case 3. (M. H. 99465) Twelve days prior to her death, this 75 year old woman complained of a stiff neck. This symptom was followed by coma of 36 hours duration. Grossly bloody spinal fluid was obtained by spinal tap. Stupor succeeded coma, and she was admitted to Montefiore Hospital eight days prior to death. The patient was completely areflexic and presented neither nuchal rigidity nor Kernig's sign. Though she had a history of hypertension, the admission blood pressure was 160 mm. Hg systolic and 65 mm. diastolic. The pupils were small but equal and reacted to light. She responded to pinprick bilaterally, but less vigorously on the left, and there was suggestive left facial paresis of central type. An electroencephalogram showed very slow activity from all leads. Carotid angiography disclosed a small aneurysm at the termination of the right internal carotid artery in the region of the posterior communicating artery. There was no shift of the major cerebral arteries. Four days before death, the patient became comatose. The left extremities did not respond to noxious stimuli. Nuchal rigidity was noted on the following day. She remained in coma until death.

Postmortem examination: The general autopsy revealed generalized arteriosclerosis and bilateral bronchopneumonia. There was external evidence of softening of the lateral aspect of the right frontal lobe. The subarachnoid space contained blood. Severe arteriosclerosis was noted in the arteries at the base of the brain and the left internal carotid artery was narrowed. A saccular aneurysm, 6 mm. in greatest diameter, was attached by a wide neck to the internal carotid artery at its junction with the posterior communicating artery. The aneurysm was directed dorso-laterally to the latter vessel. It showed a small point of rupture on its lateral surface near its neck. The wall of the aneurysm contained atheromatous plaques. Coronal sections of the brain showed an extensive hemorrhagic infarct of the right hemisphere, involving particularly the inferior frontal gyrus and frontal operculum. Although the common carotid arteries and the entire course of the internal carotid arteries were narrowed by arteriosclerosis, there was no point of actual occlusion.

Case 4. (M. H. 101231) This 59 year old woman was well until four weeks prior to admission when she experienced a sudden pain in her neck and head. Shortly thereafter she complained of generalized headache, stiffness of the neck, nausea and vomiting. These symptoms subsided, but, on the following day, there developed ptosis of the right eyelid and paralysis of movement of the right eye. She complained of right periorbital pain. Pain and ophthalmoplegia persisted to the time of hospitalization. Admission examination revealed complete paralysis of the right third cranial nerve. The blood pressure was 180 mm. Hg systolic and 100 mm. diastolic. There were no other abnormal physical signs. No cranial bruit was heard, the fundi were unremarkable and the neck was supple. A spinal tap was performed on the morning of the second hospital day. The spinal fluid was clear, colorless and under normal pressure. Twenty minutes after completion of the procedure the patient complained of "shooting" pain over the right eye, developed a tonic seizure and lapsed into coma. Examination now disclosed generalized hyperreflexia, bilateral Babinski signs and ankle clonus. There was nuchal rigidity. The patient expired within four hours of development of these signs.

Postmortem examination: The general autopsy findings were unremarkable. The

heart was not enlarged and there was only minimal atheroma. There was a subdural hematoma on the right side as well as diffuse, fresh subarachnoid hemorrhage. The antero-medial aspect of the right temporal lobe, including the uncus and the hippocampal gyrus, was slightly softened and was covered by a layer of blood clot. The major vessels at the base of the brain formed a complete circle of Willis and showed a few scattered foci of atheroma. A large cylindrical aneurysm, measuring 3 x 1.5 cm., occupied the anterior two-thirds of the right posterior communicating artery. It appeared to originate from the internal carotid artery. There was a point of rupture, 1 cm. in widest diameter, on the posterior extremity of the aneurysmal sac. The right oculomotor nerve was thin and the right optic tract appeared depressed and flattened.

Case 5. (M. H. 60064) This 60 year old woman had had two episodes of sudden loss of consciousness accompanied by lethargy, confusion and bloody spinal fluid. The first episode occurred six months prior to admission, the second four months later. Examination at this institution showed a mild right hemiparesis and absent right superficial abdominal reflexes. She was normotensive. There was no nuchal rigidity and the spinal fluid was clear. An arteriogram showed a serpentine aneurysm of the left internal carotid artery originating just proximal to the bifurcation. The left common carotid artery and the left internal carotid artery were ligated in the neck in separate stages. Following these procedures, the patient had no recurrence of subarachnoid hemorrhage. She died three years and four months post-operatively because of generalized metastases from a primary malignant melanoma of the finger.

Postmortem examination: The left fourth finger had been amputated. Metastases from a malignant melanoma were widely disseminated in the general organs. There were many metastases to the dura and brain. The configuration of the circle of Willis was normal. A serpentine aneurysmal dilatation was seen in the left posterior communicating artery and arose at the junction of this vessel with the internal carotid artery. It measured 7 mm. in greatest diameter. On section, the wall of the aneurysm was markedly fibrotic. Its lumen was occluded by an organized, recanalized and partially hyalinized thrombus. Both intracranial internal carotid arteries were patent.

GROUP 2

Case 6. (M. H. 63558) This 11 year old girl was known to have congenital heart disease. Nevertheless, she had been comparatively free of symptoms and required no cardiac medication. Eight hours before admission she suddenly developed severe headache and had a generalized convulsion. On admission she was lethargic and uncooperative. Nuchal rigidity was prominent. The right pupil was larger than the left and reacted poorly to light. Funduscopy revealed bilateral papilledema and hemorrhage in the vicinity of the right disc. There was a left Babinski sign and generalized symmetric hyperreflexia. In the course of the first hospital day, the patient developed a right oculomotor nerve paralysis and a left hemiparesis. An electroencephalogram showed diffuse slow activity without focal predominance. A large aneurysm near the internal carotid-posterior communicating artery junction was visualized by right carotid angiography. On bed rest the patient's condition improved until the sixth hospital day when she suddenly lapsed into coma with a dilated, fixed right pupil. Lumbar puncture was performed and yielded grossly bloody spinal fluid under a pressure of more than 600 mm. H_2O. A Selverstone clamp was applied to the right internal carotid artery in the neck. However, the patient expired on the following day.

Postmortem examination: The general autopsy disclosed multiple congenital car-

diovascular anomalies including cor triloculare, transposition of the aorta and pulmonary artery, patent ductus arteriosus, coarctation of the aorta (infantile type) and subinfundibular aortic stenosis. The cerebral leptomeninges were diffusely stained by blood (Figure 1). There was a layer of clotted blood at the base of the brain filling the interpeduncular fossa and the cerebellopontine angles. There was slight herniation of the right cingulate gyrus and severe herniation of the cerebellar tonsils. The right uncus was partially destroyed. Under the floor of the third ventricle was a 2 cm. mass of firm clotted blood, partially enclosed by a thin aneurysmal sac. The aneurysm, which was widely ruptured on its ventrolateral aspect, took origin from the right internal carotid artery

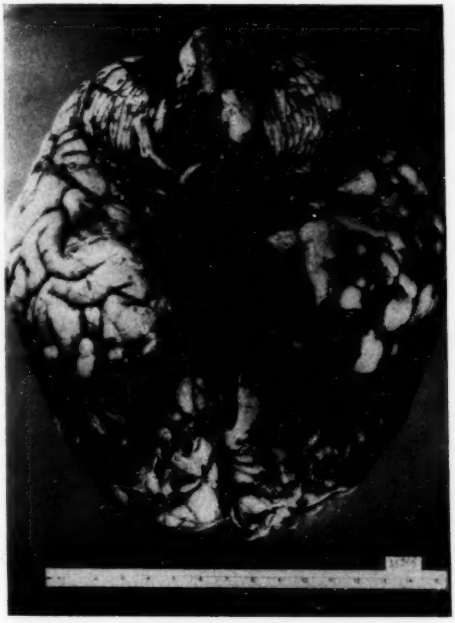


FIG. 1. Case 6. There is massive subarachnoid hemorrhage at the base of the brain. The aneurysm was situated at the junction of the right internal carotid and posterior communicating arteries, but is covered. A large clot near the right uncus indicates the point of rupture into the temporal horn.

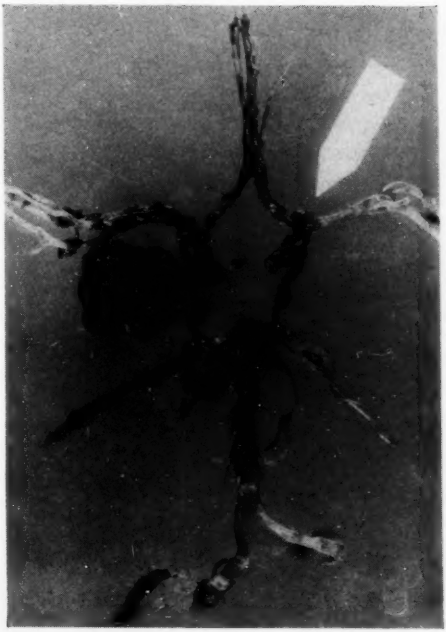


FIG. 2. Case 6. The large ruptured aneurysm arises from the right internal carotid artery and is directed posteriorly parallel to the posterior communicating artery. The arrow indicates a small unruptured aneurysm of the left anterior and middle cerebral arteries.

1 cm. proximal to its bifurcation. The aneurysm had a long, narrow neck and pointed posteriorly in a plane parallel to the posterior communicating artery. The aneurysmal sac and its contained blood compressed the right optic nerve, while the third nerves were severely compressed and flattened. There was a second aneurysm at the bifurcation of the right internal carotid artery. It was thin-walled, intact, measured 3 mm. and protruded anteriorly (Figure 2). The other major arteries were normal and the circle of Willis was complete. On coronal section the entire ventricular system, except for the left temporal and occipital horns, was filled by a fresh, jelly-like, deep red blood clot which had ruptured through the septum pellucidum. The clot in the sac of the aneurysm communicated with the

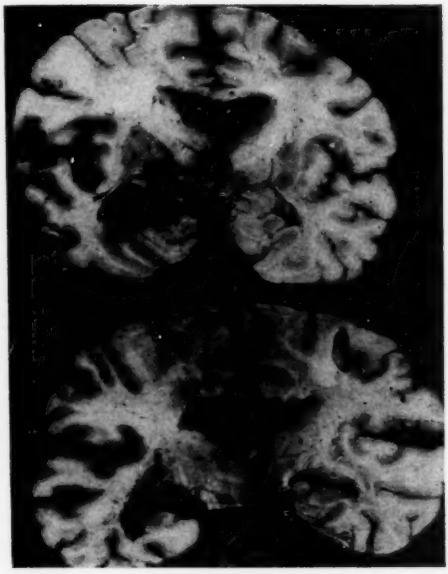


FIG. 3. Case 6. The right temporal horn and the bodies of the lateral ventricles are filled by blood. The septum pellucidum is torn. The aneurysm has been removed.

conspicuously dilated, blood-filled right temporal horn by a defect in the partially destroyed right uncus (Figure 3). An area of hemorrhagic infarction was present in the right putamen. The Sylvian aqueduct and the fourth ventricle were occluded by a cast of blood. There was a 5 mm. zone of hemorrhage in the pontine tegmentum.

Case 7. (M. H. 100540) A 65 year old woman was admitted in deep coma three hours following a "stroke." She had a history of hypertension with systolic readings as high as 250 mm. Hg. Her neck and extremities were completely flaccid. The left pupil was fixed and of pinpoint size, while the right pupil was obscured by a corneal opacification. The patient expired eight hours after admission.

Postmortem examination: Generalized atherosclerosis, left ventricular hypertrophy and mild nephrosclerosis were found in the general autopsy. The cerebral leptomeninges were diffusely stained by dark red blood clot. Bilateral cerebral swelling, right greater than left, was evidenced by flattened gyri and narrowed sulci. The right cingulate gyrus and both cerebellar tonsils were herniated. A thick layer of blood clot covered the ventral surface of the brain, including the brain stem. The ventro-medial aspect of the right temporal lobe, including the uncus and hippocampal and fusiform gyri, was the site of a deep, blood-filled cavity which penetrated into the temporal horn of the right lateral ventricle. The circle of Willis was complete, and the major arteries showed only occasional, small atheromatous plaques. An aneurysm, 8 x 5 mm. in widest diameter, was found arising from the junction of the right internal carotid and posterior communicating arteries. It was attached by a broad base and pointed posteriorly. Its postero-superior aspect presented a pinpoint site of rupture with pouting edges. The cranial nerves were completely buried in blood clot. On coronal section the area of rupture into the right temporal horn was located at the level of the mammillary bodies. A cast of blood greatly expanded the right temporal horn and the ventricular system was filled by blood except for the left temporal horn. There were numerous petechiae in the pontine tegmentum.

Case 8. (O. S. 77) This 40 year old man had a history of proteinuria and moderate elevation of the blood pressure. A clinical diagnosis of polycystic kidney disease had been made. His father had died of polycystic kidney disease and a sister was said to have the same condition. The patient was generally free of symptoms, however, until ten hours prior to his admission to another hospital when he awoke with severe headache followed by disturbance of vision, vomiting, and a generalized convulsion. On hospitalization he was noted to be lethargic. He complained of severe headache, and showed nuchal rigidity and a large hemorrhage in the left fundus. The blood pressure was 170 mm. Hg systolic and 100 mm. diastolic. Grossly bloody fluid under increased pressure was found on lumbar puncture.

Coma and right hemiparesis developed 11 days after admission. Angiography revealed an aneurysm of the left internal carotid artery. The left common carotid artery was ligated in the neck. However, the patient's condition deteriorated progressively from the fifth postoperative day. Decerebrate rigidity developed and he expired on the 19th postoperative day, one month after admission.

Postmortem examination: The general autopsy confirmed the presence of polycystic kidney disease. Examination of the brain revealed findings similar to Cases 6 and 7. There was an aneurysm arising from the area of junction of the left internal carotid and posterior communicating arteries. Blood had ruptured through the left hippocampal gyrus into the left temporal horn.

Case 9. (M. H. 18028) This 27 year old man presented symptoms and signs of chronic rheumatic heart disease complicated by bacterial endocarditis. There were symptoms of emboli to the visceral organs. The patient had repeated episodes of subarachnoid hemorrhage, one of which appeared to be the immediate cause of death.

Postmortem examination: The general autopsy confirmed the presence of bacterial endocarditis. The mitral and aortic valves were involved by a healed rheumatic endocarditis and there was cardiac hypertrophy and dilatation. The brain showed findings similar to previous cases in this group. The left temporal horn was enormously dilated by blood clot. A ruptured mycotic aneurysm was demonstrated in histologic sections, but its exact location was not described.

Case 10. (M. H. 74536) This 51 year old woman was well until 45 days prior to her death when she suddenly became "dizzy," fell to the floor and had a left-sided clonic seizure followed by unconsciousness. On recovering consciousness she complained of severe headache. Lumbar puncture disclosed an initial pressure of 340 mm. H_2O and the spinal fluid was grossly bloody. There were no lateralizing neurological signs. The patient improved slowly following the initial episode, but two further attacks of subarachnoid bleeding occurred 20 days and 10 days before death. She was admitted in coma two hours prior to her demise when she showed nuchal rigidity, papilledema and bilateral Babinski signs. The blood pressure was 150 mm. Hg systolic and 60 mm. diastolic. The only lateralizing sign was a right hyperreflexia.

Postmortem examination: The general organs showed no noteworthy abnormality. In particular there was only minimal arteriosclerosis. The under surface of each leaflet of the cerebral dura was covered by a dark brown neomembrane which was thicker on the left side. The cerebral leptomeninges were stained brown diffusely. The gyri were flattened, while the unci, especially the left uncus, and the left cingulate gyrus were herniated. There was slight coning of the cerebellar tonsils. The orbital surface of the left frontal lobe in the region of the gyrus rectus was disrupted and replaced by blood clot. The circle of Willis was complete and the vessels were free from atheromatous plaques. A 5 mm. aneurysmal sac protruded superiorly and medially from the left internal carotid artery and was adherent to the left optic nerve. There was no thrombus in the aneurysmal sac. It invaginated the olfactory sulcus and, from this point, a hemorrhagic tract penetrated the left frontal lobe (Figure 4). The limb of the left forceps minor of the corpus callosum, the medial portion of the head of the caudate nucleus, and adjacent putamen and internal capsule were destroyed by hemorrhage. Blood filled the left lateral ventricle and the third and fourth ventricles. There was no pontine hemorrhage.

GROUP 3

Case 11. (M. H. 55931) A normotensive 37 year old woman awoke with severe headache and pain in the neck 19 days prior to death. Except for an episode of headache two months previously, the past history

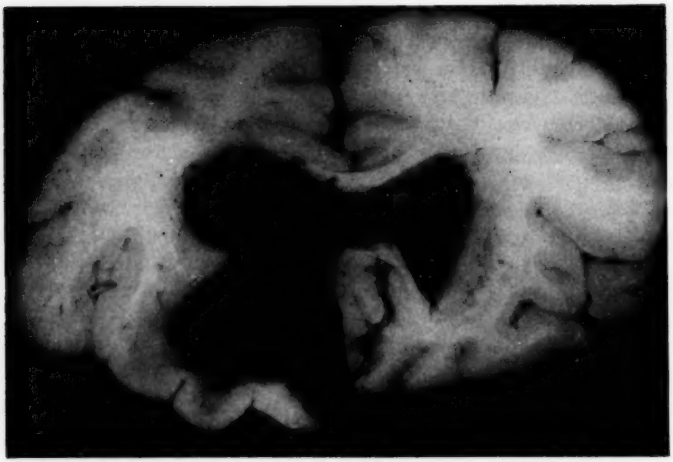


FIG. 4. Case 10. Blood has ruptured through the gyrus rectus into the frontal white matter and anterior horn.

was unremarkable. Her complaints worsened progressively. Four days from their onset she became confused and on the fifth day, bloody spinal fluid was obtained by lumbar puncture. On the seventh day from onset of symptoms she developed a left facial weakness. A gradually progressive paresis of the left arm then appeared and was followed by left hemiplegia. Finally, she became comatose. A spinal tap two days prior to death showed clear fluid and a pressure of 160 mm. H_2O. The neck at this time was slightly resistant to flexion. An electroencephalogram revealed diffuse slow-wave activity in the delta range with a right frontal predominance. Ankle clonus and bilateral Babinski signs were noted on the day before death.

Postmortem examination: The general autopsy did not disclose any abnormality. External examination of the brain revealed a small amount of subarachnoid blood in the area of the right Sylvian fissure and in the basal cisterns. There was a moderate degree of cerebral swelling with bilateral uncal and tonsillar herniations and herniation of the right cingulate gyrus. The major arteries were free of atheroma. A large aneurysm, 1 cm. in greatest diameter, arose from the right middle cerebral artery at the bifurcation of the right internal carotid. The Sylvian fissure contained a large hematoma which dissected into the bordering gray and white matter of the frontal lobe for a short distance (Figure 5). The hematoma measured 4 x 3 cm. A shift of the ventricular system from right to left and two small infarcts of the right centrum semiovale were other findings. There was no blood in the ventricular system. A large hemorrhage was noted in the pontine tegmentum.

Case 12. (M. H. 59109) This 38 year old woman had had right-sided headache and focal seizures involving the left limbs for six years. Repeated examinations during this time revealed mental deterioration and bilateral Babinski signs. The only focal sign was a slight flattening of the left nasolabial fold. Repeated lumbar punctures with spinal fluid analysis were normal. Twelve days prior to death she developed signs of subarachnoid hemorrhage and was admitted to Montefiore Hospital. She presented nuchal rigidity and a bloody spinal fluid under increased pressure. The blood

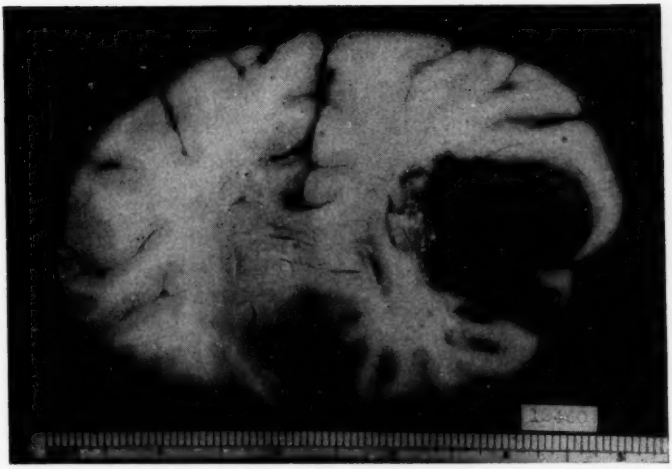


FIG. 5. Case 11. The hematoma in the Sylvian fissure has penetrated into the frontal lobe. A ruptured aneurysm of the right middle cerebral artery has been removed.

pressure was 150 mm. Hg systolic and 90 mm. diastolic. She expired following a series of convulsions.

Postmortem examination: The general autopsy did not disclose evidence of arteriosclerosis. There was a double renal pelvis with double ureter on the right. On examination of the brain a thin film of subarachnoid blood was noted covering the right Sylvian fissure and the ventral surface of the brain stem. There was swelling of the right hemisphere, and the right uncus and cerebellar tonsils were herniated. Cerebral arteries were free from atheromatous change. An aneurysm was found in the right Sylvian fissure. It arose from the right middle cerebral artery and measured 1 cm. in greatest diameter. A large hematoma, measuring 5 cm. in greatest diameter, was present in the right Sylvian fissure and compressed and infiltrated adjacent frontal and temporal cortex and white matter (Figure 6). The ventricular system was shifted from right to left but had not been penetrated by the hemorrhage (Figure 7). There was no blood in the ventricles. The left putamen contained an infarct, 1.2 cm. in widest diameter. There were a few pontine hemorrhages.

Case 13. (M. H. 67732) This 60 year old woman had a history of hypertension. Nine years before death she had an episode of transient left facial weakness, and numbness and paresthesias of the left hand. Two months prior to death she had severe headache, nuchal rigidity and vomiting for five days. Neurological examination then showed a hemorrhage in the right retina, hyperactive tendon reflexes on the left and a left Babinski sign. Bloody fluid was obtained at lumbar puncture. Electroencephalography demonstrated generalized slowing with predominance of abnormality in leads from the right temporal area. Her symptoms improved gradually. About three hours before death she had a convulsive seizure and lapsed into coma with dilated, fixed pupils and bilateral Babinski signs. The blood pressure was 164 mm. Hg systolic and 90 mm. diastolic. She died within five minutes of admission to this institution.

Postmortem examination: Examination of the general organs revealed only minimal systemic arteriosclerosis. The findings in the brain were similar to those of Cases 11

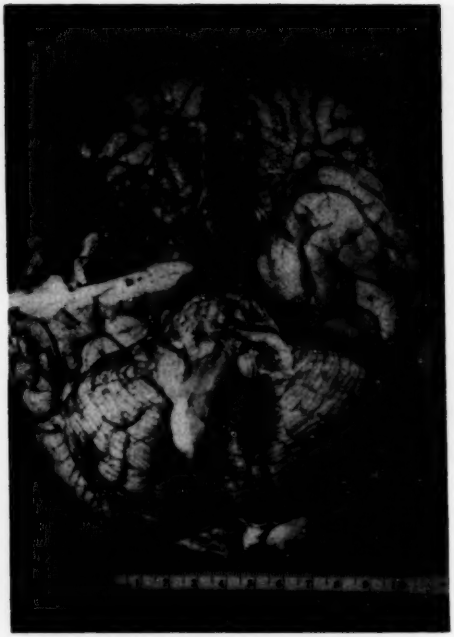


FIG. 6. Case 12. There is a hematoma in the right Sylvian fissure. A ruptured aneurysm of the right middle cerebral artery is obscured from view by blood clot.

and 12. A moderate amount of subarachnoid blood overlay the right Sylvian fissure. An aneurysm, 7 mm. in greatest diameter, arose from the right middle cerebral artery at the site of its first major bifurcation (Figure 8). A friable thrombus was loosely attached to the superior aspect of the aneurysm. There was moderate atherosclerosis of the major arteries at the base of the brain. The Sylvian fissure on the side of the aneurysm was markedly expanded by blood which had extended for a short distance into the right frontal centrum semiovale. There was a conspicuous shift of the ventricular system from right to left. The right lateral ventricle contained a minimal amount of blood.

Case 14. (M. H. 86716) Twenty-four hours prior to admission this 49 year old man suddenly developed weakness of the left hand which rapidly progressed to left hemiparesis. He had a known history of rheumatic heart disease. Admission neurological examination disclosed left facial weakness of central type, left hemiparesis and a left Babinski sign. There was no

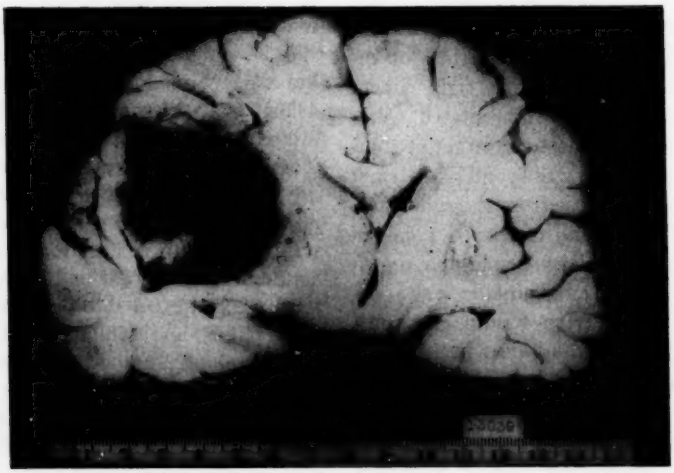


FIG. 7. Case 12. A hematoma in the right Sylvian fissure has extended into adjacent white matter. The right uncus is herniated and the lateral ventricles are displaced to the left.

FIG. 8. Case 13. Note the ruptured aneurysm of the right middle cerebral artery at the first bifurcation within the Sylvian fissure.

nuchal rigidity but a bilateral Kernig's sign was reported. Lumbar puncture revealed blood-stained spinal fluid and an initial pressure of 180 mm. H_2O. A diagnosis of subacute bacterial endocarditis was made on clinical and laboratory evidence. A ruptured cerebral mycotic aneurysm was suspected. Despite massive antibiotic therapy the patient expired on the eighth hospital day. The patient's neck was reported to be supple on repeated examination.

Postmortem examination: General autopsy findings included healed rheumatic mitral valvulitis, left ventricular hypertrophy and subacute bacterial endocarditis involving the mitral valve. Of note was the finding of congenital absence of the right kidney and ureter. On examination of the brain a saccular aneurysm was discovered at the junction of the right internal carotid and middle cerebral arteries. It measured 1 cm. in greatest diameter and its inferior surface was necrotic and ruptured. The right Sylvian fissure was filled with blood. The orbital and lateral surfaces of the right frontal lobe also exhibited subarachnoid hemorrhage. The right island of Reil, adjacent to the hemorrhage in the Sylvian fissure, was partly destroyed. The ventricular system was free of blood, but was shifted from right to left. There were petechiae in the pontine tegmentum.

Case 15. (M. H. 28911) A 53 year old man with known rheumatic heart disease was admitted acutely ill. There was clinical evidence of subacute bacterial endocarditis and congestive heart failure. He was uremic. During the final three days of illness he had repeated convulsions. Slight left-sided hyperreflexia was noted at this time. Subarachnoid hemorrhage was not suspected clinically.

Postmortem examination: The general autopsy findings were those of chronic rheumatic heart disease complicated by bacterial endocarditis and multiple septic emboli. The brain showed abnormalities similar to those of Case 13. A ruptured mycotic aneurysm was found in the right middle cerebral artery within the Sylvian fissure. The Sylvian fissure contained a large hematoma, 5 x 5 cm. in widest diameter. The hemorrhage had extended into the frontal white matter.

Case 16. (M. H. 85757) This 57 year old hypertensive man had a mild head injury without loss of consciousness six days prior to admission. Three days before admission he had a generalized seizure. On recovery he complained of severe, persistent headache. On hospitalization neurological examination revealed minimal left-sided hyperreflexia and slight weakness of dorsiflexion of the left foot. The remainder of the neurological examination was normal. In particular there were no signs of meningeal irritation and the fundi were normal. The blood pressure was 180 mm. Hg systolic and 100 mm. diastolic. Lumbar puncture revealed clear fluid under normal pressure. Microscopic examination of the spinal fluid disclosed 42,000 crenated red blood cells per cu. mm.

Carotid arteriography showed an aneurysm of the right middle cerebral artery at the point of the first bifurcation. There were also angiographic signs indicative of a mass in the right temporal lobe. On the third hospital day a right fronto-temporal craniotomy was performed with evacuation of a hematoma from the region of the anterior part of the temporal lobe. The hematoma was believed to be intracerebral. The neck of the aneurysm was clipped. The patient did comparatively well during the first three postoperative days. He was alert, but showed a monoparesis involving the left upper limb. His condition then deteriorated. He lapsed into stupor with signs of a left hemiparesis. He was reexplored, but there was no evidence of subdural hemorrhage or intracerebral hematoma. The bone flap was sacrificed for decompression. The patient improved temporarily following the second procedure, but then lapsed once more into stupor and periodically exhibited a decerebrate state. He was taken once more to the operating room where an attempt at ventricular puncture was unsuccessful. An air study was performed from below but filling was not obtained above the level of the tentorium. Accordingly, further attempts at ventriculography were made and a needle was ultimately inserted in the right lateral ventricle. A ventriculogram was now obtained and showed marked shifting of the ventricular system to the right. The left subdural space was tapped through the burr hole and a large amount of yellow fluid was removed. Radiographs following this removal indicated shift of the ventricular system back to the midline. However, there was no clinical improvement and the patient expired on the 38th hospital day.

Postmortem examination: There was no noteworthy abnormality of the general organs. There was only a mild degree of systemic arteriosclerosis and the heart was not enlarged. The brain showed diffuse flattening of the cerebral gyri. The leptomeninges about the right Sylvian fissure were stained by blood. The right temporal lobe in the vicinity of the Sylvian fissure showed foci of laceration and necrosis which had been produced at surgery. Both unci were herniated. The major vessels at the base of the brain were normal. A clipped, 7 mm. aneurysm arose from the right middle cerebral artery 3 cm. distal to its point of origin. The aneurysm had a broad base. No point of rupture could be demonstrated. The right middle cerebral artery and its branches were patent. There were multiple hemorrhagic infarcts of that part of the right temporal lobe adjoining the fissure of Sylvius. A large hematoma, such as seen in the previous cases, was not present in this instance. Presumably, it had been removed at surgery.

Case 17. (M. H. 98506) This 31 year old man had known polycystic kidney disease and hypertension. Fourteen days prior to admission there was sudden onset of headache which persisted. A day before admission he suddenly lapsed into coma. Examination following hospitalization showed stupor, right hemiparesis with hyperreflexia and central facial weakness, and a right Babinski sign. Several hemorrhages were

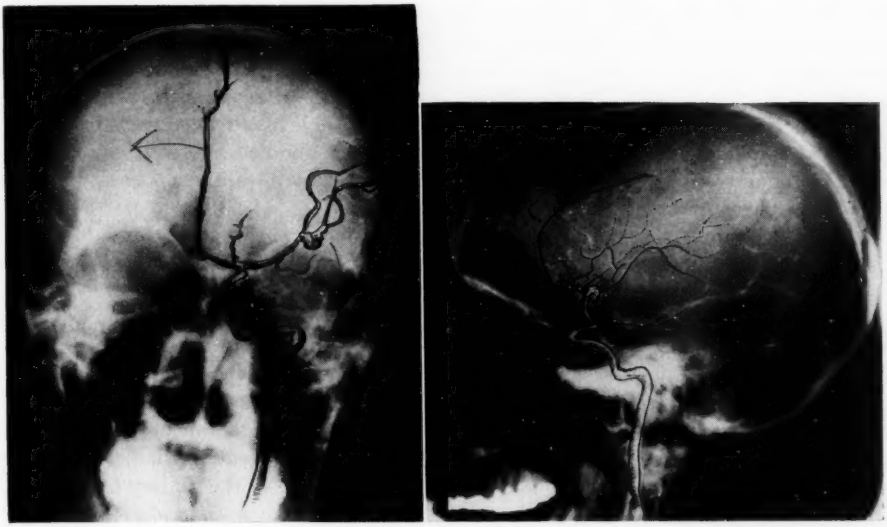


FIG. 9. Case 17. A left carotid angiogram shows an aneurysm of the middle cerebral artery. Because of a Sylvian hematoma, the anterior and middle cerebral arteries are displaced.

seen on funduscopy and there was moderate nuchal rigidity. The blood pressure was 175 mm. Hg systolic and 100 mm. diastolic. Lumbar puncture yielded evenly blood-tinged spinal fluid under a pressure of 380 mm. H_2O. Carotid angiography demonstrated an aneurysm at the first bifurcation of the left middle cerebral artery. This artery was greatly elevated and the anterior cerebral artery was bowed to the opposite side (Figure 9). A diagnosis of a hematoma within the left temporal lobe was made and the patient was operated upon the day of admission. A left temporal lobe hematoma was evacuated and the neck of a bilobed aneurysm of the left middle cerebral artery was clipped. The patient's sensorium and right hemiparesis improved following surgery. The nuchal rigidity disappeared. However, 13 days after operation pyuria and hematuria developed. Signs of a severe pyelonephritis appeared and the patient died in uremia on the 44th hospital day.

Postmortem examination: The general autopsy confirmed the clinical diagnosis of congenital polycystic disease of the kidneys. In addition the right kidney was the site of an acute pyelonephritis with abscess formation, and there was a massive hematoma in the upper pole. The liver contained congenital cysts. The heart was not enlarged and the systemic arteries showed only minimal atheroma. The brain did not exhibit signs of increased intracranial pressure and there were no pontine hemorrhages. The major vessels forming the circle of Willis were elastic and thin-walled, but the configuration was anomalous. Both anterior cerebral arteries arose from the right internal carotid artery by a single stem. A thin, transversely running vessel connected the anterior cerebrals with the left middle cerebral artery. The left posterior cerebral artery arose from the ipsilateral internal carotid and was connected to the basilar artery by a vessel about 2 mm. in diameter. The antero-lateral portion of the ventral surface of the left frontal lobe and the tip of the left temporal lobe were necrotic. Arising from the left middle cerebral artery 2 cm. from its point of origin was an aneurysm containing numerous

metallic clips and measuring 1 x 1 cm. The aneurysmal sac was very thin and originated by a broad neck from a point of branching of the middle cerebral artery. There was no gross atheromatous change. A point of actual rupture could not be identified. Coronal sections through the cerebral hemispheres revealed hemorrhagic softening of the frontal, temporal and parietal lobes adjacent to the left fissure of Sylvius. There was no evidence of an intracerebral hemorrhage, nor was there a hematoma within the Sylvian fissure. The latter was presumably present prior to operation and had been removed surgically. The ventricular system was normally disposed and was free of blood clot.

DISCUSSION

The age of these patients ranged from 11 to 75 years. Noteworthy was the relative absence of atherosclerosis of the cerebral vessels. The aneurysms shared in this freedom from severe atheroma, with the single exception of a specimen from a 75 year old hypertensive patient (Case 3). A small atheromatous plaque was commonly found near or on the aneurysmal sac, but a causal relationship between atheroma and aneurysm was never demonstrable.

An anomaly of the circle of Willis was found in one instance (Case 16). In five cases congenital anomalies were present in other organs. There were multiple cardiovascular anomalies in Case 6, polycystic kidneys in Cases 8 and 17, congenital absence of one kidney in Case 14, and a congenital double renal pelvis and ureter in Case 12. The anomalies in Cases 12 and 14 were on the same side as the cerebral aneurysm. On three occasions ruptured mycotic aneurysms were demonstrated (Cases 9, 14 and 15), and were the direct cause of death.

The locations of the aneurysms were as follows: 1) the main trunk of the supraclinoid portion of the internal carotid artery between the point of exit from the cavernous sinus and the major bifurcation (two cases from Group 1 and two from Group 2); 2) the internal carotid-posterior communicating artery junction (three cases from Group 1 and two cases from Group 2); 3) the middle cerebral artery within the Sylvian fissure (all seven cases of Group 3).

A summary of observations distinguishing the three groups is presented in Table 1.

Except for Case 6, the aneurysms were solitary. In Case 6 there was a small unruptured aneurysm of the middle cerebral artery on the opposite side. Save for Cases 4 and 6 none of the aneurysms were more than 1 cm. in greatest diameter. In these exceptional cases the widest diameter was 3 cm. and 2 cm. respectively.

With few exceptions the aneurysmal sac clearly originated from a site of bifurcation and presented a rounded shape and wide neck. However, in Case 6, an aneurysm arising from the internal carotid artery below the major bifurcation presented a long, narrow neck which ran parallel to the posterior communicating artery.

Five of seven aneurysms of the middle cerebral artery were located on the right side. This predominance of right-sided involvement has been noted by others. Of 107 cases of middle cerebral artery aneurysms included in five reports (2, 6, 9, 11, 12), 72 were located on the right.

The direction of the long axis of the aneurysm varied with the site of origin. When the posterior communicating artery was implicated, the aneurysm pointed laterally and posteriorly. Aneurysms arising from the main trunk of the internal carotid artery were directed medially. Therefore, aneurysms at the former site were usually adherent to the oculomotor nerve and closely applied to the ipsilateral temporal lobe, while aneurysms of the internal carotid artery were commonly closely attached to the ipsilateral optic nerve and chiasm. Visual symptoms are known to be

TABLE 1

Summary of Characteristics of Groups Discussed in Text

	Group 1	Group 2*	Group 3
Number of cases	5	4	7
Subarachnoid blood	++++	+++	+
Blood in the Sylvian fissure	+	+	++++ Unilateral
Intraventricular blood	0	++++	0
Site of rupture into the ventricle	0	Vicinity of uncus	0
Angiographic appearance of Sylvian vessels	Normal	Elevated	Severely elevated; aneurysm of Sylvian group.
Lateral ventricles	Narrowed but normally disposed.	Unilateral dilatation, particularly temporal horn.	Flattened and severely shifted.
Origin of aneurysm	Main trunk of internal carotid, or posterior communicating-internal carotid junction.	Usually, junction of posterior communicating and internal carotid arteries.	Middle cerebral artery and its Sylvian branches.

* Case 10, in which the hemorrhage ruptured into the left frontal horn, is not included in the table.

common in aneurysms originating from either site (6, 9), taking the form of oculomotor paralysis when the posterior communicating-carotid junction is involved and giving rise to loss of acuity or field defects when the main stem of the internal carotid is implicated. Middle cerebral artery aneurysms were directed laterally parallel to the normal direction of blood flow in this vessel.

Group 1: In all five cases there were clinical indications of meningeal irritation, as evidenced by nuchal rigidity and bilateral Kernig signs, and of increased intracranial pressure. Multiple episodes of subarachnoid hemorrhage were constant. Lateralizing signs were clearly present in two patients (Cases 1 and 3) and were mistakenly attributed by the clinician to intracerebral extension of the subarachnoid bleeding. At necropsy intracerebral hemorrhage was not demonstrated in these patients, but the frontal operculum and the dorsal part of the island of Reil on the side of the aneurysm were involved by fresh hemorrhagic infarction. Neither occlusion of an artery nor formation of a thrombus in an aneurysmal sac was demonstrated in association with the infarcts. Similar cases have been noted by others (7, 9), and cerebral arterial spasm, occurring at the time of aneurysmal rupture, has been invoked in explanation.

Angiography demonstrated the presence of the aneurysm in each case. In none was displacement of the anterior or middle cerebral arteries found. It might also be mentioned in connection with the question of arterial spasm discussed in the previous paragraph, that angiography never disclosed focal narrowing of the vessel at the site of the aneurysm nor impairment of filling distal to it.

In three patients ligation of the common carotid artery was performed. Death occurred within 16 hours of surgery in Case 1 and within five days in Case 2. In both the patient's condition was grave at the time of surgery and pontine hemorrhages were present at autopsy. In Case 4 there was no recurrence of subarachnoid hemorrhage after operation. Neurological signs disappeared and the patient remained well for more than three years only to succumb to a metastatic malignant melanoma. At

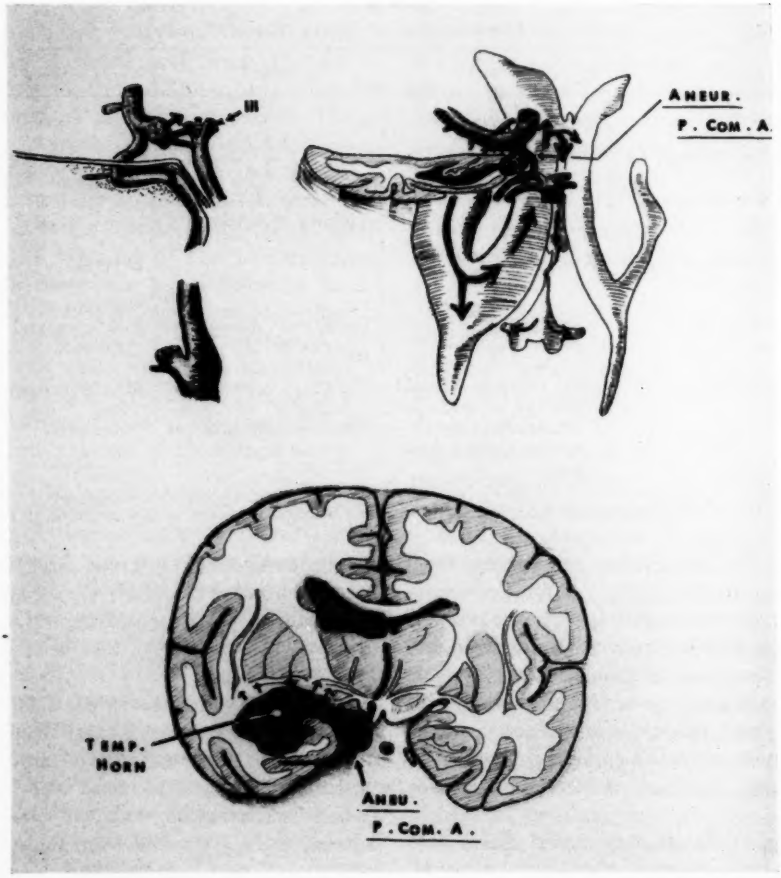


FIG. 10. A drawing shows the mechanism of rupture into the temporal horn in Group 2 aneurysms. The aneurysm is depicted as arising from the junction of the internal carotid and posterior communicating arteries.

autopsy in this case the aneurysm was completely occluded by an organized thrombus.

Group 2: Five cases comprised this group. The characteristic lesion was the destruction by hemorrhage of the hippocampal gyrus, uncus and fimbria hippocampi with penetration into the temporal horn (Figure 10). Because of severe damage to the hippocampus and dentate gyrus, prominent uncal herniation was produced and might have been a factor in the development of signs of oculomotor nerve compression in Case 6. Cases similar to those included in this group have been illustrated by Blackwood (1; Fig. 266), Globus (3; Case 10), Hamby (4; Case 40) and Richardson and Hyland (8; Case 6). The temporal horn and ipsilateral lateral ventricle were distended by the intraventricular hemorrhage, which sometimes broke through the temporal horn and involved structures inferior to the lenticular nucleus, or even spread to the pallidum and internal capsule. A cast of blood extended through the lateral and third and fourth ventricles, and presented at the foramina of Luschka. A considerable

amount of subarachnoid blood was always present in the basal cisterns and extended forward into the Sylvian fissures.

Clinically, all cases of this group presented nuchal rigidity and signs of increased intracranial pressure. Involvement of the internal capsule by extension of hemorrhage beyond the confines of the temporal horn led to contralateral long tract signs in Cases 6 and 8. Differential diagnosis between Group 1 and Group 2 was provided by carotid angiography which showed elevation of the middle cerebral artery, with some degree of shift of the anterior cerebral to the opposite side. Only one patient, Case 6, was treated surgically. The internal carotid artery was occluded in the neck by a Selverstone clamp. The patient was in grave condition at the time of operation and expired within a day. Necropsy showed recent hemorrhages into the pontine tegmentum.

Case 10, although somewhat exceptional, belongs to this group. The aneurysm arose from the main trunk of the internal carotid artery, an infrequent origin for aneurysms causing intracerebral hemorrhage, and was adherent to the brain surface in the region of the olfactory sulcus. Rupture led to hemorrhage through the gyrus rectus into the white matter of the frontal lobe, and thence through the forceps minor into the frontal horn. There was also a subdural extravasation of blood, although there was little subarachnoid hemorrhage. The pattern of intraventricular rupture resembled the Type II anterior communicating artery aneurysm described by Hirano and coworkers (5). Case 2 of Robertson (9) was similar to the authors' Case 10 except that there was no subdural hematoma and the ruptured aneurysm arose from the site of the major bifurcation of the internal carotid artery.

Group 3: There were two constant characteristics of the cases belonging to this group. First, the origin of the aneurysm was from the middle cerebral artery within the Sylvian fissure. Second, rupture led to formation of a massive hematoma largely localized to the leptomeninges within the Sylvian fissure. During development of the hematoma, the fissure of Sylvius was expanded and the surrounding brain tissue was compressed. The ventricular system was displaced to the opposite side and the ipsilateral ventricle was narrowed (Figure 11). On external examination subarachnoid blood was usually largely limited to the vicinity of the Sylvian fissure on the side of the aneurysm, although there was some staining of the leptomeninges on the ventral surfaces of the frontal lobe and brain stem. In the five non-operated cases the Sylvian hematoma dissected into the cortex of the island of Reil and operculum, and into underlying white matter, but the hemorrhagic penetration was of limited extent and in no case reached the ventricular system. Pontine hemorrhages frequently accompanied the supratentorial hematoma. The Sylvian hematomas produced by ruptured aneurysms of the Sylvian group have been previously described as examples of intracerebral hemorrhage (1, 3, 4, 6, 7, 8, 9). Admittedly, ruptured aneurysms of the middle cerebral artery and its branches within the Sylvian fissure may cause uncomplicated, diffuse subarachnoid hemorrhage (7, 12). Or, as in Robertson's Cases 1 and 13, rupture may lead directly to intracerebral hemorrhage with little leakage of blood into the meninges. In most instances, however, ruptured aneurysms of the Sylvian group have resulted in the picture described in this paper.

Clinically, as might be expected from the pathologic findings, all patients in this group had lateralizing signs on neurological examination. Unlike Groups 1 and 2, nuchal rigidity was not always prominent, or was even lacking. The spinal fluid was usually bloodstained, rather than frankly bloody. In Case 11 the spinal fluid was normal at a time when the patient was comatose. In some patients the relatively slow develop-

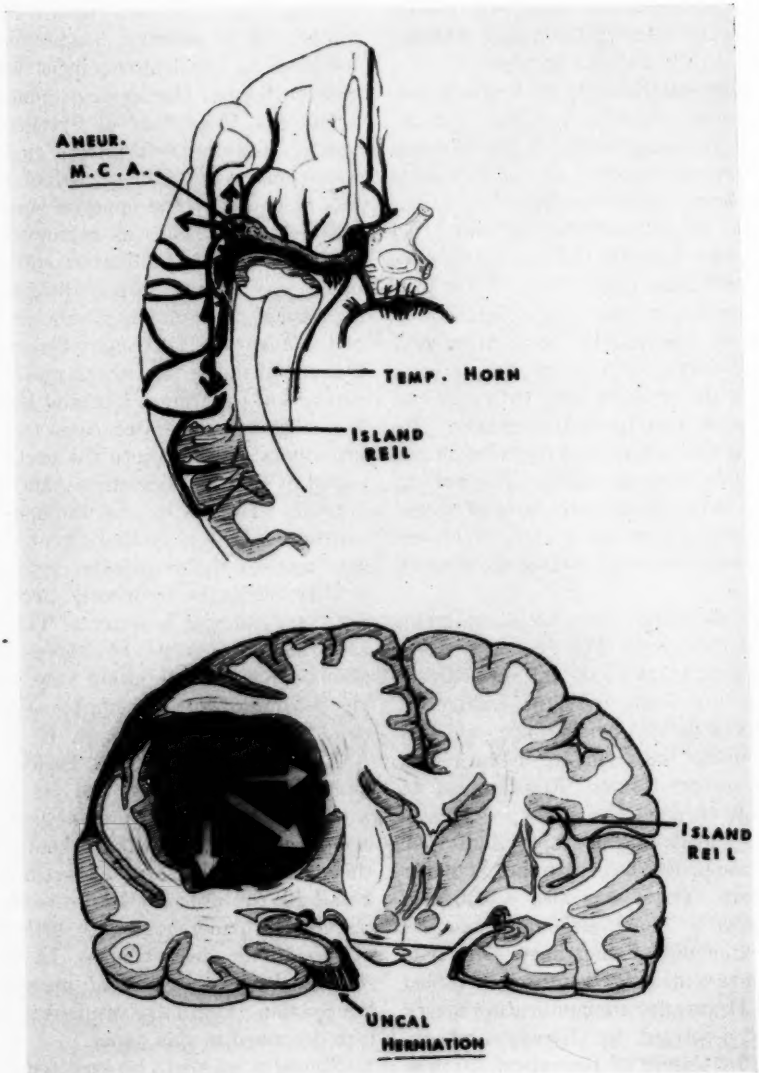


FIG. 11. This is a drawing of a hematoma within the Sylvian fissure in a Group 3 case. The ruptured aneurysm was located on the middle cerebral artery.

ment of focal signs, the presence of seizures and the relative subtlety of the evidences of meningeal irritation may suggest a cerebral neoplasm, while cerebral thrombosis or spontaneous intracerebral hemorrhage may be suspected in others. Complaints of severe persistent headache and neck pain, the absence of hypertension, the preservation of consciousness and the presence of blood in the spinal fluid were all features which, individually or collectively, were of clinical differential diagnostic im-

portance. Carotid angiography was performed in Cases 16 and 17 and demonstrated the radiologic abnormalities which are pathognomonic for this group, namely: 1) evidence of an avascular mass lesion in the middle fossa; 2) an aneurysm of the middle cerebral artery.

An operation was performed in Cases 16 and 17, and consisted of evacuation of blood clot from the area of the temporal lobe with application of clips to the ruptured aneurysm. Necropsy examination of the brains in these cases did not reveal evidence of displacement of the ventricular system and there were no hemorrhages into the tegmentum of the brain stem. In Case 17 there was an excellent response to operation, but the patient died from renal failure. The pathologic anatomy of cases of Group 3 would suggest that good results may be obtained from intracranial surgery.

SUMMARY

Three pathologic groups were delineated in 17 patients with ruptured aneurysms of the supraclinoid internal carotid and middle cerebral arteries. In the first group, bleeding was confined to the subarachnoid space. The second group was marked by diffuse subarachnoid hemorrhage and by disruption of brain tissue with penetration of the bleeding into the ventricular system. The usual route of rupture into the ventricular system was through the rostral hippocampal cortex of the temporal lobe in the vicinity of the uncus. Typically, cases of the second group were associated with aneurysms arising at the junction of the internal carotid and posterior communicating arteries. The third group was characterized anatomically by formation of a large hematoma within the Sylvian fissure. The brain and contained ventricular system were displaced and compressed, but hemorrhage did not penetrate into the latter. Cases of the third group were caused by aneurysms of the Sylvian arteries. Clinical and radiological findings were presented which may allow clinical differentiation of each group.

REFERENCES

1. Blackwood, W. Vascular disease of the central nervous system. In *Neuropathology*. E. Arnold Ltd., London, 1958.
2. Dandy, W. E. *Intracranial arterial aneurysms*. Comstock Publishing Co., Ithaca, 1944.
3. Globus, J. H. Cerebral aneurysm and massive nontraumatic cerebral hemorrhage. J. Neuropath. & Exper. Neurol., **2**: 365–391, 1943.
4. Hamby, W. B. *Intracranial Aneurysms*. C. C Thomas, Springfield, 1952.
5. Hirano, A., Terry, R. D. and Zimmerman, H. M. Ruptured aneurysms of the anterior communicating artery. J. Nerv. & Ment. Dis., **128**: 309–322, 1959.
6. Frankel, K. and Alpers, B. J. The clinical syndrome of aneurysm of the middle cerebral artery. A. M. A. Arch. Neurol. & Psychiat., **74**: 46–67, 1955.
7. Meadows, S. P. Intracranial aneurysms. In *Modern Trends in Neurology*, Feiling, A. ed., pp. 391–465. P. B. Hoeber, New York, 1951.
8. Richardson, J. C. and Hyland, H. H. Intracranial aneurysms: clinical and pathological study of subarachnoid and intracerebral hemorrhage caused by berry aneurysms. Medicine, **20**: 1–83, 1941.
9. Robertson, E. G. Cerebral lesions due to intracranial aneurysms. Brain, **72**: 150–185, 1949.
10. Walsh, F. B. *Clinical Ophthalmology*. 2nd ed. Williams & Wilkins, Baltimore, 1957.
11. Walton, J. N. *Subarachnoid Haemorrhage*. E. S. Livingstone Ltd., Edinburgh, 1956.
12. Williams, R. R., Bahn, R. C. and Sayre, G. P. Congenital cerebral aneurysms. Proc. Staff Meet. Mayo Clin., **30**: 161–168, 1955.

A NEW DRUG CAUSING SYMPTOMS OF SENSORY DEPRIVATION

Neurological, Electroencephalographic and Pharmalogical Effects of Sernyl

JOHN S. MEYER, M.D.,[1] F. GREIFENSTEIN, M.D. and M. DEVAULT, M.D.

During the clinical evaluation of the compound 1-(phenylcyclohexyl) piperidine, monohydrochloride (Figure 1) named Sernyl® for possible therapeutic use by the Departments of Neurology and Anesthesiology

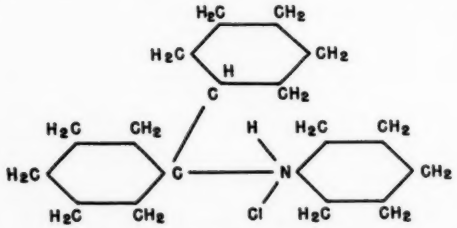


Fig. 1. Structural formula of Sernyl.

of Wayne State University, College of Medicine it was noted that administration of the drug occasionally resulted in symptoms similar to those observed in individuals subjected to sensory deprivation (1, 3, 5). As judged by our clinical observations in man this drug is presumed to have a relatively selective action on sensory cortex, thalamus and midbrain resulting in demonstrable impairment of pain, touch, proprioception and discriminative aspects of sensation. Motor function is unimpaired until high doses are administered, when ataxia and nystagmus result. Subjects in whom sensory deprivation has been induced by Sernyl regularly report feelings of anxiety, depression or fear together with difficulty in thinking and concentration. In higher dosage illusional, delusional and hallucinatory experience and a sensation of displacement commonly occur.

Because of the potential importance of these phenomena in relation to the physiological effects of perceptual isolation and the potential usefulness of this drug in experimental psychology and psychiatry, it is considered appropriate to report our observations.

Material and Methods

One hundred and two patients have been observed during and after administration of Sernyl. Eighty patients received the drug intravenously while lying prone on a table in a quiet operating room prior to various surgical procedures. In some of them neurological examinations were made before, during and after administration of the drug together with concurrent records of electroencephalogram, blood pressure, respirations and electrocardiogram. Electroencephalograms were recorded using bipolar technique with an 8 channel type 3D Grass electroencephalograph. Ten conventional electrode placements were made on the scalp. Blood pressure was recorded through an arterial canula inserted into the brachial artery and connected to a Statham transducer. Blood pressure and electrocardiogram were recorded with a Sanborn polygraph. Respirations were measured with the subjects breathing room air through a one-way nonrebreathing valve. The expired gases were collected in a self-recording Tissot spirometer. The remaining 22 patients suffered from neurological disorders and were given Sernyl orally in various doses to evaluate the possible usefulness of the drug in thalamic pain, atypical facial pain, postherpetic neuralgia, post-encephalitic Park-

[1] Departments of Neurology and Anesthesiology, College of Medicine, Wayne State University, and the Detroit Receiving Hospital, Detroit, Michigan. This work was aided by a grant from Parke, Davis and Co., who made Sernyl® available for clinical investigation.

inson's disease, paralysis agitans, Huntington's chorea, rubral tremor, benign familial tremor and double athetosis. Of the 80 subjects who received large doses of the drug intravenously, thirteen developed a confusional psychosis of 12–72 hours duration characterized by feelings of unreality, depression, anxiety and delusional and illusional experience. One patient who received a larger dose than the others had a perceptual disorder that persisted for four days. In some of these subjects observations of behavior and neurological examination were made during the psychotic episode and were compared with the subject's memory of the experience after the drug had been excreted and behavior had returned to normal.

Of the 22 subjects administered oral preparation of the drug for neurological disorders, eight showed evidence of impaired mentation. None of these subjects had any impairment of mentation prior to the administration of the drug but one patient with benign familial tremor was blind due to glaucoma. All the patients who received the drug by mouth were ambulatory and were observed on the ward or in the outpatient department.

RESULTS

NEUROLOGICAL OBSERVATIONS

Intravenous administration of Sernyl regularly produced significant sensory change after 7.5 mgs. of the drug had been administered. Pin-prick was no longer appreciated as painful and response to all forms of sensory stimulation was either appreciably slowed or diminished. After receiving 8 mgs. of the drug one patient was unable to read the time or recognize a painful stimulus, but was able to identify a packet of cigarettes and a key, this very slowly. When 9.5 mgs. of the drug had been administered, vertical nystagmus and bilateral ptosis commonly occurred. After 10.5 mgs. drowsiness appeared and common objects could not be identified by vision or touch, although motor movement was preserved. In higher dosage ptosis, vertical and horizontal nystagmus, dysarthria and crowing respiration were common and the corneal and pupillary light reflexes became depressed. At this stage there was no response to any form of stimulation, but spontaneous movements of the extremities often occurred. The tendon jerks became increased and the plantar reflexes could not be elicited. The visual fields were demonstrated to be intact although recognition of objects was impaired. At this stage minor surgical procedures could be carried out without discomfort.

After recovery from intravenous injection of 7 mgs. of Sernyl, patients were able to recall events clearly. At higher dosage objects seemed to "float away" and the subjects had the sensation that the body went numb and was displaced. The sensation of displacement of the body in relation to the environment was consistently noted by those patients receiving more than 7 mgs. of Sernyl by intravenous injection. After 10 mgs. hallucinatory experiences were commonly noted which appeared to be patterned according to the personality structure and past behavioral experience of the subject. Many of our patients with strong religious backgrounds reported that "God was taking them away" or that they were being "carried up into the clouds." Rarely were feelings of persecution reported.

CONFUSIONAL PSYCHOSIS

Several patients were examined who remained confused for 12–96 hours after the injection of the drug. The confusional state was characterized by feelings of unreality, depersonalization, persecution, depression and intense anxiety. Some became agitated and anxious. During these states of confusional psychosis defects in sensory perception were demonstrable. Pin-prick sensation was regularly impaired and a partial visual agnosia was occasionally demonstrable. In many instances objects were falsely interpreted and the stimuli responsible for an il-

lusional reaction could be identified. There was also disorientation to time and place. For example, one patient of stoical and cooperative personality structure was being observed in the neurological ward with a diagnosis of pituitary tumor. Carotid angiography was performed under intravenous Sernyl anesthesia. Several hours after the patient had been returned to the ward he became restless and noisy attempting to leave the ward and making accusatory remarks to the nurses and attendants. When he had recovered he explained that he recalled the intravenous drip being started, then his body became numb and he was carried up into the clouds where a group of guards were shouting orders (there were orders being made by the surgeon to the radiology staff during the procedure) then the "guards" attempted to transfix his chest with a massive "spike" (probably the arterial needle). After his recovery the patient explained that during this period of psychotic behavior he was under the impression that "the guards were trying to capture and kill me."

The most prolonged state of confusional psychosis we have seen persisted for four days. The subject was a 45 year old woman who received 0.3 mg. of Sernyl per kilogram of body weight for anesthesia during cholecystectomy. On recovery from anesthesia she was confused, disoriented and failed to recognize people; she tended to lapse into sleep if left alone. She required catheterization because her bladder became distended without discomfort. She talked to relatives who were not present and visual hallucinations occurred. She responded when called by name and stated that she was willing to get out of bed, but felt sleepy. Speech was slightly slurred. She did not know where she was because she was "all mixed up." She recognized the side-boards on her bed, but believed that she was at home. She could not recognize her family because "I cannot think." She stated that the date was 1898 and she had virtually no recent memory at all. When asked her name she gave her maiden name and could not recall her husband's name. She knew her age, the color of a lemon, but not the color of an orange. She was unable to read the time on a clock face. Simple calculation was grossly impaired and she could not read or write. She obeyed simple motor commands with ease. She was unable to recognize a flower. The visual fields were intact since she responded to threats by blinking, but she could not identify common objects presented to her. Nystagmus was present and there was no response to pin prick over the entire body. Although motor strength was entirely normal, position sense was poor and vibration sense was absent. Seven days after operation, neurological and mental examination were entirely normal and she was ambulatory.

Subjects who received the drug by mouth developed milder degrees of mental disorder which were investigated in detail. Two ambulatory and working patients who were being treated with 7.5 mgs. of Sernyl daily for pain, reported relief of pain but inability to think or solve problems. For example, one of them who was an engineer complained that he sat looking at blueprints all day, but was unable to solve a simple problem that usually required thirty minute's work. Another subject treated with Sernyl for postherpetic neuralgia reported that an oral dose of 12.5 mgs. daily abolishd her pain, but in addition produced a feeling as though she were "dying," a "sinking sensation." She said that all her "senses" disappeared and she had no feeling. Her thinking became void; and objects appeared smaller than normal. The beds in the ward appeared to be "as small as marbles" and looked as though they were "stuck to the ceiling." In her case, everything seemed displaced upward and she was displaced downward. Her entire body was numb and as her "senses" came back her hands "looked square." This situation lasted for 3–4 hours and during this time pin-prick sensation was demon-

strably absent, but she could identify coins by palpation, slowly but correctly. Two-point discrimination was also slowly, but correctly perceived.

Subjects with post-encephalitic Parkinsonism tolerated the drug very poorly and became withdrawn, dull and apathetic. Presumably, this decreased tolerance to the drug is due to already existing damage to midbrain and thalamus by the disease process itself. One patient who received 7.5 mgs. daily for six weeks became semi-stuporous and catatonic. His limbs would persist if placed in abnormal postures for several minutes. When the drug was discontinued recovery was prompt. Another patient with post-encephalitic Parkinsonism, receiving 7.5 mgs. daily, reported that this produced a "novocaine" sensation over her entire body and she seemed to be floating in air. It is of interest that patients with paralysis agitans do not appear to show the same degree of sensitivity to the drug, and tremor has been reduced or abolished in several patients.

There also appears to be a lowered threshold to the drug in blind individuals. A colored patient with benign familial tremor and blindness due to glaucoma was treated for seven days with 7.5 mgs. of Sernyl daily, without improvement of his tremor. On the seventh day this previously quiet and placid individual became delusional with a strikingly religious flavor. He began clapping his hands in time to non-existent banjo-music and saying, "Brother, let me tell you how I've been saved." When the drug was discontinued these symptoms rapidly cleared.

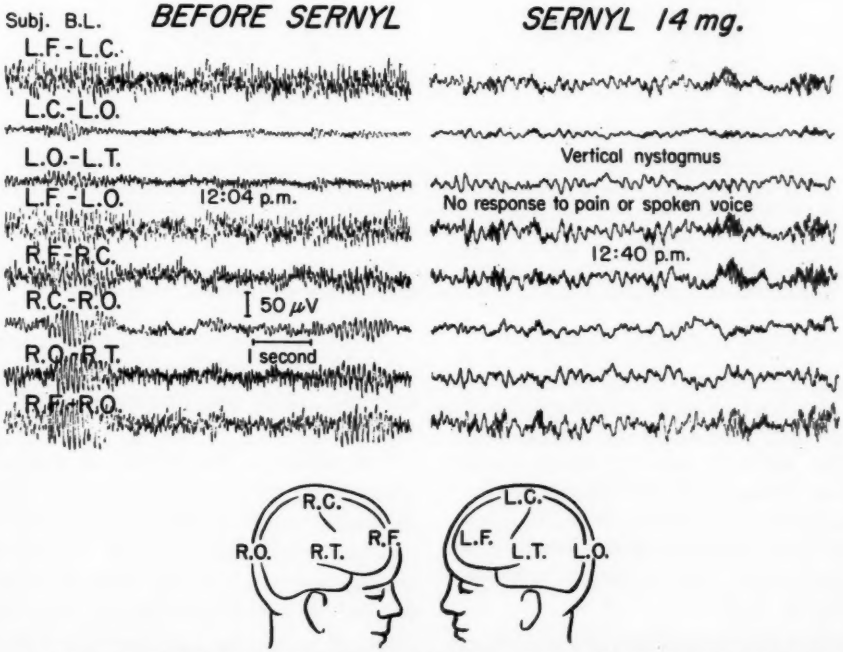


FIG. 2. Samples of continuous EEG recorded from a 49 year old woman weighing 170 lbs. while receiving Sernyl intravenously prior to excision of a lipoma of the left arm. In the record to left, prior to the administration of Sernyl there is alpha activity except in the frontal regions where there is beta rhythm. After 14 mgs. of Sernyl there is no response to pain or spoken voice and vertical nystagmus is present. The EEG shows diffuse slowing in the theta and delta bands.

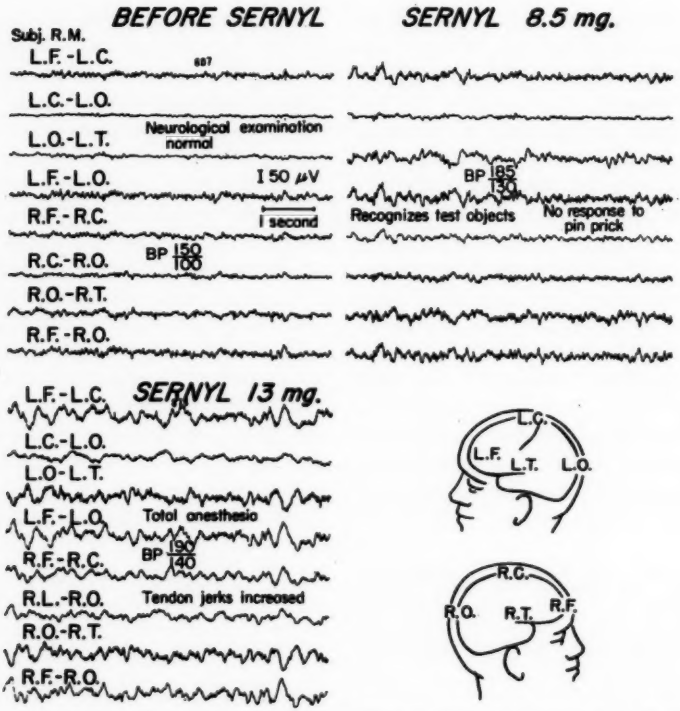


FIG. 3. Samples of continuous EEG recorded from a 33 year old woman while receiving Sernyl intravenously prior to diagnostic curettage of the uterus for menometrorhagia. Before Sernyl the record shows low voltage beta activity. After 8.5 mgs. of Sernyl the BP is elevated and there is no response to pin-prick, but she can identify visual test objects. There is slowing in all regions except the central regions.

After 13 mgs. of Sernyl there is high voltage showing in the delta range and there is no response to all types of stimulation, the blood pressure and tendon-jerks are increased.

ELECTROENCEPHALOGRAPHIC CHANGES

After intravenous administration of 7.5 mgs. of Sernyl, changes consistently appear in the EEG. First there is a disappearance of alpha and beta activity and the appearance of slowing in the theta range in all regions except the central ones. In higher dosage theta and delta slowing becomes diffuse and there is an increase in amplitude (Figures 2, 3, 4).

CHANGES IN BLOOD PRESSURE, RESPIRATION AND HEART RATE

The effects of Sernyl on blood pressure, respiration, and the electrocardiogram were studied on seven volunteer subjects.

Sernyl was administered intravenously in a 0.1% solution at the rate of 5 c.c. per minute until a total dose of 0.25 mgm./kg. had been given.

The effect on respiration is shown in Table 1. As the blood concentration of the drug is increased, the minute volume of respiration is increased. At 0.25 mg./kg. the average increase is of the order of 1140 ± 907 cc. for the seven subjects. Rate and depth of respiration both increase with increasing minute volume, although these changes also appear to be small. At 0.25 mg./kg., six of the seven subjects exhibited a tidal volume equal to or greater than the

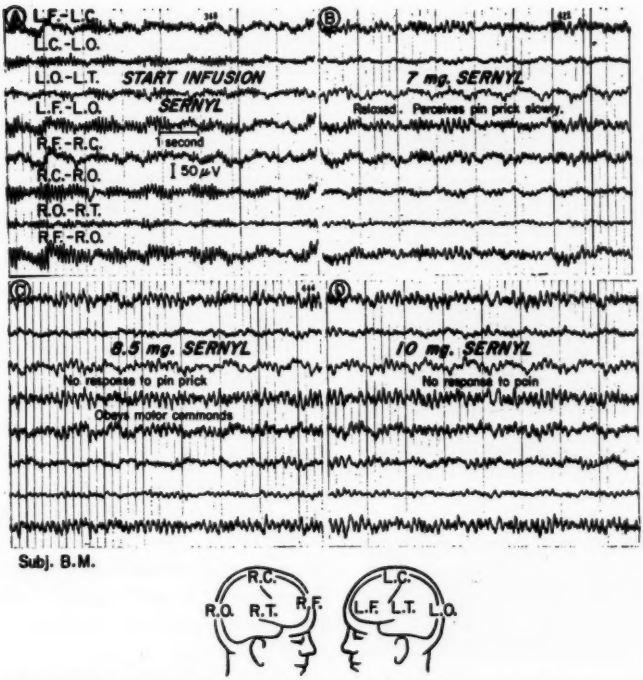


FIG. 4. Samples of a continuous electroencephalographic record made on a patient while receiving Sernyl intravenously. A. Prior to infusion there is alpha activity. B. After 7 mgs. slowing appears and pain sense is diminished. C. and D. shows progressive changes in EEG at higher dosage, which were never marked.

TABLE 1

Respiratory Response to Sernyl

Mg./kg.	0.06	1.125	0.19	0.25
Min. Vol.	140 ± 407	640 ± 407	640 ± 957	1140 ± 907*
Tidal Vol.	16.7 ± 39.5	27.1 ± 50	27.4 ± 66.9	15.3 ± 52.9
Resp. Rate	1.86 ± 1.81	1.43 ± 2.61	3.43 ± 1.79	2.57 ± 3.82

* The ± deviations represent confidence values and not standard deviation from the mean. (P = 0.01)

TABLE 2

Circulatory Response to Sernyl—mm. hg. Change from Normal

Mg./kg.	0.06	0.125	0.19	0.25
Systolic B.P.	7.9 ± 6.07	23 ± 14.4	26 ± 9.8	25.7 ± 12.9*
Diastolic B.P.	7.43 ± 4.43	18.6 ± 9.3	17.7 ± 4.6	19 ± 5
Pulse rate	0.33 ± 4.68	8 ± 13.2	7.8 ± 8.4	5.1 ± 14.9

* The ± deviations represent confidence values and not standard deviation from the mean (P = 0.01)

control value, and five of seven an increase in respiratory rate.

Circulatory changes are shown in Table 2 and a typical tracing representative of the blood pressure changes are depicted in Figure 5. Both diastolic and systolic blood pressure increased slightly over the control values until approximately half the total dose had been given. At this point a plateau was reached and the blood pressure maintained at this new level for approximately thirty minutes.

Pulse rate changes were not as consistent.

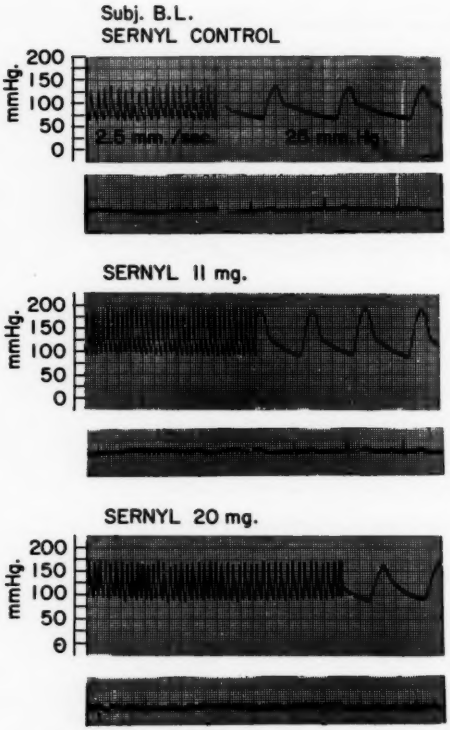


FIG. 5. Records of blood pressure and electrocardiogram before Sernyl (top trace) after 11 mg. (middle trace) and after 20 mg. (bottom trace) of Sernyl intravenously. (See text.)

Four of seven subjects showed a slight increase in pulse rate, while the remaining three exhibited a mild bradycardia after 0.25 mg./kg. had been given.

The electrocardiogram exhibited no arrhythmias or other abnormal patterns, although changes in the EKG have been demonstrated in lower species.

DISCUSSION

It appears from our data that Sernyl produces a form of sensory deprivation due to the action of the drug on the central nervous system interfering with sensory perception. The type of sensory loss and the associated changes in cranial nerves, blood pressure and reflexes suggest that the drug acts primarily on the sensory cortex, brain-stem and thalamus. This concept is further supported by the fact that subjects with disorders that are known to affect the brain-stem and thalamus (such as post-encephalitic Parkinsonism) show a marked sensitivity to the drug. In addition we have treated four cases of thalamic pain with the drug, with partial or complete relief of pain. The drug does not affect rubral tremor (one subject) or the involuntary movements of Huntington's chorea (five patients). In a few patients with paralysis agitans and in one case of double athetosis the involuntary movements were improved, but care must be taken in dosage to avoid the unpleasant side effects of sensory deprivation. Similarly, patients suffering from post-herpetic neuralgia, atypical facial pain due to syringomyelia were afforded relief, but often found themselves unable to think clearly and became anxious or depressed. Toxic effects on bone marrow, liver and kidney have not been noted in any of our patients or in experimental animals (2, 4).

After higher doses of Sernyl a deficit in sensation is demonstrable and symptoms appear that are similar to those reported during sensory deprivation on normal volunteers (1, 3, 5). We have seen similar symptoms in patients with porphyric and post-infectious polyneuritis who have been treated in a tank-type respirator for prolonged periods of time, in individuals immobilized with Crutchfield tongs for treatment of cervical fracture and in immobilized and temporarily blinded patients following ophthalmological surgery. The concept that the "psychotic states" produced by Sernyl are related to the phenomenon of sensory deprivation is further supported by the fact that in one of our patients who was blind, symptoms of confusional psychosis were produced by doses of Sernyl which did not produce symptoms in individuals with intact vision.

SUMMARY

Neurological, electroencephalographic and pharmacological data have been presented

on over 100 individuals who have been treated with Sernyl, a drug that appears to exert a blocking action on the thalamus and midbrain. This drug eventually produces a blocking of all forms of sensation with pain sensation disappearing first. Motor movement is not impaired, except for ataxia, until the patient becomes unconscious. Blood pressure and reflexes are increased and vertical nystagmus appear in high dosage of the drug. Early and sometimes prolonged symptoms appear that are similar to those reported in sensory deprivation studies; namely, anxiety, illusional, delusional and hallucinatory phenomena with a feeling of displacement. Interference with thinking processes is an early complaint. It is concluded that Sernyl produces a "centrally mediated" sensory-deprivation syndrome.

REFERENCES

1. Bexton, W. H., Heron, W. and Scott, T. H. Effect of decreased variation in the sensory environment. Canad. J. Psychol., **8:** 70–76, 1954.
2. Chen, G. Pharmacological studies of Sernyl. Personal Communication.
3. Solomon, P. Sensory deprivation and the human mind. Research Reviews, Office of Naval Research, **4:** 8–11, 1958.
4. Weston, J. K., Weston, K., Fisken, R. A. and Rentner, F. Toxicological studies of Sernyl. Personal Communication.
5. Wexler, D., Mendelson, J., Leiderman, H. and Solomon, P. Sensory deprivation: A technique of studying psychiatric aspects of stress. Arch. Neurol. & Psychiat., **79:** 225–233, 1958.

BLOOD AND URINARY SEROTONIN AND 5-HYDROXYINDOLE ACETIC ACID LEVELS IN SCHIZOPHRENIC PATIENTS AND NORMAL SUBJECTS[1]

AARON FELDSTEIN, PH.D.,[2] HUDSON HOAGLAND, PH.D.[2] AND HARRY FREEMAN, M.D.[3]

Woolley (16, 17) first proposed early in 1954 that a deficiency or excess of serotonin in the brain might be causally related to schizophrenia. If this hypothesis were correct, then schizophrenia might be reflected in high or low blood serotonin levels, or by high or low urinary levels of 5-hydroxyindoleacetic acid (5-HIAA), a metabolite of serotonin. We wish to report our findings on blood serotonin and urinary 5-HIAA in chronic and acute schizophrenic patients and in normal, *i.e.* non-psychotic subjects.

All blood serotonin analyses were performed by the fluorometric procedure of Udenfriend and co-workers (14). A study of two females and 15 male normal subjects, members of the Worcester Foundation staff, indicated that the mean concentration of serotonin in blood was 0.19 μg/ml, S.D. ± 0.08, S.E.M. ± 0.02, range from 0.06 to 0.39 μg/ml. The results are in agreement with the findings of Udenfriend and co-workers (15) who reported that the serotonin levels in normal subjects ranged from 0.1 to 0.3 μg/ml. A study of 22 male chronic schizophrenic patients selected from the research ward of the Worcester State Hospital indicated a mean of 0.17 μg/ml, S.D. ± 0.06, S.E.M. ± 0.01, range from 0.06 to 0.31 μg/ml. The *t*-test showed that the difference between the means of the schizophrenic patients and the normal subjects was not significant ($p > 0.3$).

A study of two female and 13 male acute psychotic patients, all admitted not more than a month before the analysis, indicated a blood serotonin mean value of 0.12 μg/ml, S.D. ± 0.07, S.E.M. ± 0.02, range from 0.05 to 0.29 μg/ml. Statistical analysis indicated that the difference between the means of this group and the normal group was significant at the 0.02 level of confidence. The difference between the acute psychotic group and the chronic schizophrenic group also was statistically significant ($p < 0.05$; See Table 1).

All 5-HIAA measurements were made using the Udenfriend method involving extraction and colorimetric determination (13). Morning urine samples were collected from 32 male chronic schizophrenic patients; the mean of the 5-HIAA output was 225 μg/hr (5.4 mg/day), S.D. ± 85, S.E.M. ± 15, range from 113 to 438 μg/hr. Urine collections from 36 normal male subjects resulted in a mean of 203 μg/hr (4.9 mg/day), S.D. ± 76, S.E.M. ± 13, range from 47 to 471 μg/hr. The difference between the two groups was not significant ($p > 0.2$). The mean urinary 5-HIAA excretion rate of eight normal female subjects was 214 μg/hr (5.1 mg/day), S.D. ± 161, S.E.M. ± 57, range from 50 to 549 μg/hr. The results are summarized in Tables 2 and 4.

Morning urine samples were collected from 23 acute psychotic male patients. The urinary output of 5-HIAA was 229 μg/hr (5.5 mg/day), S.D. ± 166, S.E.M. ± 36, range from 33 to 638 μg/hr. The difference between this group and normal male subjects was not significant ($p > 0.05$). A study of 12 acute psychotic women indi-

[1] This work has been supported by grants from The Ford Foundation and from the Scottish Rite Committee for Research in Dementia Praecox.

[2] The Worcester Foundation for Experimental Biology, Shrewsbury, Massachusetts, and the Worcester State Hospital, Worcester, Massachusetts.

[3] Present address: Medfield State Hospital, Medfield, Massachusetts.

TABLE 1

Blood Serotonin Concentrations Measured Fluorimetrically (14) in Normal Subjects and in Psychotic Patients. The Asymmetry Estimate Was Made by the Method of Snedecor (12)

Subjects (Groups)	Subjects (No.)	Mean (μg/ml)	S.D.	S.E.M.	Median (μg/ml)	Asymmetry			Range (μg/hr)	
						g_1	t	p	Low	High
Normal Males	15	0.19	0.08	0.02	0.18	+0.93	1.60	>0.1	0.06	0.39
Females	2	0.15	0.01	0.01	0.15					
Chronic Schizophrenics										
Males	22	0.17	0.01	0.01	0.17	+0.08	0.32	>0.7	0.06	0.31
Acute Psychotics										
Males	13	0.13	0.07	0.02	0.11	+1.36	2.21	<0.02	0.05	0.29
Females	2	0.07	0.03	0.02	0.07				0.05	0.09

TABLE 2

Urinary 5-HIAA Output Measured Colorimetrically (6) in Normal Subjects and in Psychotic Patients

Subjects (Groups)	Subject (No.)	Mean (μg/hr)	S.D.	S.E.M.	Median (μg/hr)	Asymmetry			Range (μg/hr)	
						g_1	t	p	Low	High
Normal Males	36	203	76	13	194	1.37	3.48	<0.001	47	471
	35*	195	62	10	192	0.51	1.28	>0.1	47	383
Females	8	214	161	57	184	1.38	1.83	>0.05	50	549
Chronic Schizophrenic Males	32	225	85	15	223	0.58	1.40	>0.1	113	438
Acute Psychotic	23	332	428	89	200	3.59	7.46	<0.001	33	2125
Males	22*	251	180	38	194	0.80	1.64	>0.05	33	638
Females	12	276	487	141	140	3.35	5.26	<0.001	54	1808
	11*	136	69	21	138	0.72	1.09	>0.2	54	275

* The highest value in the group was omitted and the statistics recalculated.

cated a mean of 276 μg/hr (6.6 mg/day), S.D. ± 487, S.E.M. ± 141, range from 54 to 1808 μg/hr. Statistical analysis (see Tables 2 and 4) showed that the mean of this group did not differ significantly from that of normal female subjects ($p > 0.7$).

Several contradictory reports have appeared in the literature. One report (1) showed that in a study of ten schizophrenic patients and ten normal subjects, the schizophrenic group excreted more 5-HIAA (mean, 17.2 mg/day) than the normal group (mean, 7.6 mg/day). This difference was probably due to inadequate sampling since it does not agree with the findings of other investigators (2, 5, 8, 9, 11) or with the findings in the present report (see Tables 2 and 4).

Another paper (2) reported that four catatonic patients, untreated, excreted more 5-HIAA (mean, 13.8 mg/day) than 26 untreated noncatatonic schizophrenic patients (4.4 mg/day). Again, this difference was probably due to inadequate sampling since it was found by Sano (11) and in this laboratory (5) that the catatonic group did not differ from the noncatatonic group.

Still another report (6) suggested the opposite, that schizophrenic patients excreted less than normal amounts of 5-

TABLE 3

Urinary 5-HIAA Output of Normal Subjects and Untreated Chronic and Acute Psychotic Patients, Calculated from the Data of Buscaino and Stefanachi (2)

Subjects	Subjects (No.)	Mean (μg/hr)	S.D.	S.E.M.	Median (μg/hr)	Asymmetry			Range (μg/hr)	
						g_1	t	p	Low	High
Normal	18	215	105	25	181	0.06	0.12	>0.9	42	383
Chronic Schizophrenic,										
all types	30	251	197	36	192	2.90	6.80	<0.001	33	1083
noncatatonic	26	201	98	20	179	1.10	2.46	<0.02	33	450
Acute Psychotic	11	301	251	76	254	2.01	3.04	<0.01	29	854
	10	245	180	57	250	1.24	1.80	>0.05	29	658

HIAA. The paper chromatographic method used, however, could not possibly have yielded the desired information. In our laboratory, using the same technique (4), but applying 2.5 times more urine, we obtained a correlation coefficient of only 0.61 between the frequency of appearance of the 5-HIAA and the concentration of 5-HIAA determined by Udenfriend colorimetric procedure.

A study by Buscaino and Stefanachi (2) of acute psychotic and chronic schizophrenic patients indicated that the acute psychotic group exerted greater amounts of 5-HIAA (mean, 7.2 mg/day) than the chronic schizophrenic group (mean, 4.4 mg/day). Our calculations based on the data in this report showed that the difference in the means between the two groups was not significant and the difference between the means of the acute psychotic group and the normal group (5.15 mg/day) also was not significant (see Tables 3 and 4). Our own findings showed that the excretion pattern of 5-HIAA in acute psychotic patients and normal subjects did not differ significantly (see Tables 2 and 4).

TABLE 4

Probability that the Difference Between the Means of 5-HIAA Output of Two Groups is Significant, Calculated by the Method of Snedecor (12)

	Probability	
	This Paper	Buscaino and Stefanachi (2)
Normal subjects and chronic schizophrenic patients	>0.2[a]	>0.4[b]
Normal subjects and acute psychotic patients	>0.05[a]	>0.2[b]
Chronic schizophrenic and acute psychotic patients	>0.1[a]	>0.5[b]

[a] Males.
[b] Males and females.

Of two laboratories reporting on the formation of 5-HIAA from orally administered tryptophan, one group (9) indicated no increase in urinary 5-HIAA in schizophrenic patients and a significant increase in normal subjects, whereas the other group (1) reported exactly the opposite, no increase in normal subjects and a significant increase in the 5-HIAA output in schizophrenic patients. A third investigation (8), carefully controlled, showed that an increase occurs in both groups and that there is no difference between the two groups in the pattern of excretion.

We have observed a number of high urinary 5-HIAA excretion rates as high as 1316 μg/hr (31.6 mg/day) in the chronic schizophrenic group and 1808 μg/hr (43.4 mg/day) in the acute psychotic group. This deviation from normality is statistically significant and has been observed by other investigators as well (2, 11). The physiological significance of this phenomenon remains to be determined. In one study, two patients were observed for a period of five

weeks but no correlation between output of 5-HIAA and behavior was found (5).

It is possible that the contradictions which appear in the literature stem in part from the high degree of variability in the output of 5-HIAA from hour to hour and day to day in the individual, particularly in the schizophrenic individual. Consider the study of 36 normal male subjects (see Table 2); the mean was 203 μg/hr, S.E.M. ± 13 and the distribution of values significantly skewed (g = 1.37). Multiple samples, totalling 133, were obtained from most subjects, but not all. The fact is that the highest value included in this series, 471 μg/hr (11.3 mg/day) was based on a single sample. Exclusion of this one value and recalculation based on 35 normal subjects (132 samples) gave a mean of 195 μg/hr, S.E.M. ± 10 and the distribution of the values was no longer asymmetrical (g = 0.51). The effect upon the mean of these high excretion rates is even more pronounced in the acute psychotic group. In this group, only single morning urine samples were obtained. Exclusion of the highest value caused a change in the mean from 332 to 251 μg/hr in the male and from 276 to 136 μg/hr in the female population. In both the male and the female acute psychotic patients, the distributions were highly skewed, whereas exclusion of the highest values resulted in normal distribution (see Table 2). Obviously the high values have unduly influenced the mean and comparisons of the different groups based uncritically upon the mean may not be as valid as one would have expected. This can also be observed in the data of Buscaino and Stefanachi (see Table 3).

Examination of the variation in 5-HIAA output in morning urine samples, collected over a period of weeks, indicated that we are apt to find a more normal distribution of values in the normal subjects than in the schizophrenic patients (see Table 5). Three of the six schizophrenic patients showed a statistically highly skewed distribution (g = 15.86, + 2.42, + 1.89), whereas the six normal subjects showed a normal distribution. It is apparent then that such studies as these should be designed with greater emphasis on multiple samples in order accurately to determine the mean output for each individual.

The variation in output of 5-HIAA

TABLE 5

Output of 5-HIAA Measured Colorimetrically (6) in Morning Urine Samples Collected Over a Period of Weeks From Normal Subjects and Chronic Schizophrenic Patients

Individuals	Samples (No.)	Mean (μg/hr)	S.D.	S.E.M.	Median (μg/hr)	Asymmetry			Range (μg/hr)	
						g_1	t	p	Low	High
Chronic Schizophrenic										
CAR	29	494	298	55	450	+15.86	36.58	<0.001	137	1454
AKE	29	329	130	24	362	+0.56	1.29	>0.1	79	704
DUB	16	237	108	27	283	−0.44	0.78	<0.4	14	425
OCO	16	202	106	26	169	+2.42	4.30	>0.001	96	542
O'DR	8	120	52	18	129	+0.05	0.09	>0.9	50	208
KEE	11	117	117	35	75	+1.89	2.60	<0.01	8	419
Normal										
RIA	11	311	116	35	300	+0.95	1.49	>0.1	146	571
BEN	14	257	76	20	253	0	0	>0.9	133	358
BUR	20	244	72	16	237	+0.35	0.69	>0.4	129	408
BAK	18	193	100	24	164	+0.08	0.15	>0.8	62	392
HOP	20	188	80	16	187	+0.87	1.87	>0.05	29	433
HAG	21	152	62	13	146	+0.69	1.38	>0.1	54	300

TABLE 6

Output of 5-HIAA Measured Colorimetrically (6) in Samples Collected Consecutively Over a Period of Approximately 24 Hours from Chronic Schizophrenic Patients

Chronic Schizophrenic Patients	5-HIAA output (μg/24 hr)	Consecutive Samples (No.)	Mean (μg/hr)	S.D.	S.E.M.	Median (μg/hr)	Asymmetry			Range (μg/hr)	
							g_1	t	p	Low	High
ODR	3,409	12	187	100	29	160	1.34	2.11	<0.05	63	428
KEE	4,494	7	312	251	95	180	1.00	1.26	>0.2	22	765

within a 24 hour period was studied in two chronic schizophrenic patients (see Table 6). The large variation, from 22 to 765 μg/hr, in one case, emphasized the need, not only for multiple samples, but for 24 hour collections. This high degree of variation in samples collected within hours of each other may easily be a source of error in studying the formation of 5-HIAA from serotonin, 5-hydroxytryptophan and tryptophan. For example, patient ODR yielded the following: 5:15–9:00 A.M., 126 μg/hr; 9:00–9:30 A.M., 428 μg/hr; 9:30–11:40 A.M.; 140 μg/hr. Similarly, patient KEE yielded the following: 5:15–9:00 A.M., 495 μg/hr; 9:00–12:15 P.M., 765 μg/hr; 12:15–2:30 P.M., 180 μg/hr. Had we administered any of the precursors of 5-HIAA at 9:00 A.M., we might erroneously have reported too high an increase in 5-HIAA output. This type of error might be responsible for the report (2) that when serotonin is administered intramuscularly, the schizophrenic group excreted a higher percentage as 5-HIAA than did the normal group. That the variation in output is very large is dramatically shown in this report by the fact that in nine out of 50 subjects more 5-HIAA was excreted in the 24 hours preceeding the injection than in the 24 hours after the injection, even though 6.45 mg. of serotonin (free base) was administered. In four out of these nine cases, the apparent decrease in the rate of excretion of 5-HIAA occurred in the first two hours after the administration of serotonin. Careful design of this type of experiment including an adequate statistical evaluation (pairing of the values) and multiple samples from the individual as well as a large sampling of the schizophrenic population is necessary before we may conclude with certainty that chemical differences between normal subjects and schizophrenic patients are a real phenomenon.

Our blood studies indicated that the concentration of serotonin may possibly be significantly different in acute psychotic patients from that in chronic schizophrenic patients and in normal subjects (see Table 1). The values were distributed such that in the acute patients, 80 per cent of the values were below the mean of this group, 0.12 μg/ml; in the chronic schizophrenic group, 18 per cent were below 0.12 μg/ml; and in the normal group, also 18 per cent below. This apparent difference may be due to chance or to a non-specific stress phenomenon. Most certainly, the sampling is too small and too restricted to conclude that low blood serotonin concentrations are characteristic or specific of acute psychotic individuals. It should be noted, also, that the range is almost identical in the three groups, from 0.06 to 0.39 in normal subjects, from 0.06 to 0.31 in chronic schizophrenic patients, and from 0.05 to 0.29 in acute psychotic patients which adds to the ambiquity of this finding.

It is interesting to note that Reiss (10) in discussing statistics in biochemical investigation of psychotic patients cites examples of the variation of the somatic function in which a symmetrical distribution was obtained with the mean value of the schizophrenic group close to that of the

normal control group and values lower and higher than the normal control group. In our study we found that the urinary 5-HIAA distribution curve was skewed in the direction of the high values and the blood serotonin values skewed in the direction of the low values. Interpretation of this phenomenon in terms of the behavior of the schizophrenic individual is difficult.

In all of our studies, as well as those reported in the literature, the sampling has been small and all reported differences between normal subjects and psychotic patients must be considered in the light of this fact. It is important to note also that the dietary factors in our studies, as well as others, have not been controlled and the nutritional state of the psychotic individuals is an unknown factor. We believe that there is insufficient evidence to warrant the conclusion that there is either an impairment or facilitation of the 5-hydroxyindole pathway in the schizophrenic population as a whole or in the catatonic or acute psychotic group in particular. In fact, the considerable overlap of blood serotonin values and urinary 5-HIAA values, reported by all investigators, suggests that serotonin is not causally related to schizophrenia and that a metabolic defect in the 5-hydroxyindole pathway does not exist. Such differences as do exist between normal subjects and psychotic patients relating to the higher values found in the psychotic group probably are due to nutritional factors or to a non-specific stress phenomenon.

One must not, however, lose sight of the fact that a defect in serotonin metabolism related to psychoses might occur in the brain and not be detected by measurement of the relatively large body store of serotonin in the blood or of the even larger amounts of 5-HIAA excreted in the urine. The work of Costa and Aprison (3) on brain serotonin levels in men, after autopsy, may be of immense value in evaluating the hypothesis that too much or too little serotonin is causally related to schizophrenia.

SUMMARY

The mean concentration of blood serotonin in normal non-psychotic male subjects was found to be 0.19 μg/ml, in chronic schizophrenic male patients, 0.17 μg/ml, and in acute psychotic male patients, 0.12 μg/ml. The mean urinary excretion rate of 5-HIAA in normal male subjects was found to be 203 μg/hr, in chronic schizophrenic male patients, 225 μg/hr, and in acute psychotic male patients, 332 μg/hr. The results do not indicate a metabolic defect in serotonin metabolism and therefore do not support the hypothesis of a causal relationship between serotonin metabolism and acute or chronic schizophrenia.

REFERENCES

1. Banerjee, S. and Agarwal, P. S. Tryptophan-nicotinic acid metabolism in schizophrenia. J. Proc. Soc. (Exper). Biol. & Med., **97**: 657–659, 1958.
2. Buscaino, G. A., and Stefanachi, L. Urinary excretion of 5-hydroxyindoleacetic acid in psychotic and normal subjects. A.M.A. Arch. Neurol. & Psychiat., **80**: 78–85, 1958; (Contributo allo studio del metabolismo delle sostanze indoliche nelle malattie del sistema nervoso. Noto 5. Acta neurol. **13**: 156–178, 1958.)
3. Costa, E. and Aprison, M. H. Studies on the 5-hydroxytryptamine (Serotonin) content in human brain. J. Nerv. & Ment. Dis., **126**: 289–293, 1958.
4. Feldstein, A., Dibner, I. M. and Hoagland, H. Two-dimensional paper chromatography of urinary indoles in normal subjects and chronic schizophrenic patients. In *Chemical Concepts of Psychosis*, Rinkel, M., ed., pp. 204–128. McDowell, Obolensky, Inc., New York, 1958.
5. Feldstein, A., Hoagland, H., and Freeman, H. On the relationship of serotonin to schizophrenia. Science, **128**: 358, 1958.
6. Forrest, A. D. Indole excretion patterns during treatment with reserpine. J. Ment. Sc. **103**: 614, 1957.
7. Haverback, B. J., Sjoerdsma, A. and Terry, L. L. Urinary excretion of the serotonin metabolite, 5-hydroxyindoleacetic acid in various clinical conditions. New England J. Med., **255**: 270–272, 1956.
8. Kopin, I. Tryptophan loading and excretion of 5-hydroxyindoleacetic acid in normal and schizophrenic subjects. Science, **129**: 835–836, 1959.
9. Lauer, J. W., Inskip, W. M., Bernsohn, J. and Zeller, E. A. Observations on schizophrenic

patients after iproniazid and tryptophan. A.M.A. Arch. Neurol. & Psychiat, **80**: 122–130, 1958.

10. Reiss, M. Uses and abuses of statistics in biochemical investigation of psychotic patients. In *Chemical Concepts of Psychosis*, Rinkel, M., ed., pp. 411–417. McDowell, Oblensky, Inc., New York, 1958.
11. Sano, I., Kakimoto, Y., Okamoto, T., Nakajima, H. and Kudo, Y. Über 5-Oxyindollesigsaure-Ausscheidung im Urin von Schizophrenen mit Berücksichtigung des Einflusses von Chlorpromazin und Reserpin auf den Serotoninstoffwechsel. Schweiz. med. Wchnschr., **87**: 214–217, 1957.
12. Snedecor, G. W. *Statistical Methods*, pp. 80–82; 174–176. 4th ed. Iowa State College Press, Ames, Iowa, 1946.
13. Udenfriend, S., Titus, E. and Weissbach, H. The identification of 5-hydroxyindoleacetic acid in normal urine and a method for the assay. J. Biol. Chem., **216**: 499–505, 1955.
14. Udenfriend, S., Wiessback, H. and Brodie, B. B. Assay of serotonin and related metabolites, enzymes, and drugs. In *Methods of Biochemical Analysis*, Glick, D., ed., Vol. **6**, pp. 95–130. Interscience Publ., Inc., New York, 1958.
15. Udenfriend, S., Weissbash, H. and Clark, C. T. The estimation of 5-hydroxytryptamine (Serotonin) in biological tissues. J. Biol. Chem., **215**: 337–344, 1955.
16. Woolley, D. W. and Shaw, E. A biochemical and pharmacological suggestion about certain mental disorders. Proc. Nat. Acad. Sc., **40**: 228–231, 1954.
17. Woolley, D. W. and Shaw, E. Some neurophysiological aspects of serotonin. Brit. M. J., **11**: 122–126, 1954.

DIURNAL VARIATION PATTERNS OF CIRCULATING EOSINOPHIL COUNTS AND SALIVARY NA/K IN PSYCHIATRIC PATIENTS

V. A. KRAL, M.D.,[1] B. GRAD, PH.D. AND W. HUNZINGER, M.D.

The findings discussed in this report arose in the course of an investigation on the role of stress in the etiology of senile psychoses. Part of this investigation involved a comparative study of the stress tolerance of senile patients and of control subjects and as the adrenal cortex plays a central role in the reponse to stress, it was thought desirable to use tests of adrenal cortical function as part of the test battery. In normal individuals there is a regular diurnal change in the number of circulating eosinophilic leucocytes and of the sodium over potassium ratio in saliva, and both of these functions have been shown to be under adrenal cortical control. The necessary laboratory procedures are relatively simple to perform with considerable accuracy. It was, therefore, decided to use these two physiological tests in our study.

Because each is under adrenal cortical control, it was to be expected that the diurnal variation of the circulating eosinophil count and the salivary Na/K would run parallel to each other. To determine whether this does in fact occur, preliminary investigations were conducted, and in the course of these some unexpected observation were made which seemed sufficiently interesting to warrant a brief report.

MATERIAL AND METHODS

The case material used in this study consisted of 40 patients (23 men and 17 women). Their ages ranged from 15 to 67 years with a mean and standard error of 37 ± 2 years. There was no significant difference between the ages of the men (38 ± 3 years) and of the women (37 ± 3, $0.90 > P > 0.80$). All patients were free from physical disease as far as could be determined by the usual clinical examination and laboratory tests including chest x-ray and EEG.

In 22 males and 12 females, both Na/K and eosinophil determinations were made. From four additional females and one male only saliva was collected, while in one female patient only blood could be collected for the eosinophil count.

For the eosinophil counts 5 cc. of blood were taken from the veins of the arms. A phloxine-propylene glycol solution was used as diluent and the eosinophils of each person were counted directly in 4 platforms of 2 Fuchs-Rosenthal hemocytometer chambers. When repeated 0.05 ml. blood aliquots were withdrawn from the same 5 ml. sample of blood, stained and counted, the error did not exceed 5%. When repeated counts were made of any one sample in the counting chamber without repeatedly charging the chamber, the error was less than one per cent.

The method of collection of the saliva samples and the determination of their Na/K content has been described in detail elsewhere (6). It should be emphasized that only unstimulated, mixed saliva such as is normally found in the mouth between meals was collected. Subjects were watched to see that they neither ate, drank, smoked or napped for more than one-half hour prior to beginning of the test, for these factors are known to influence the salivary Na/K (6). Samples were collected at 7 A.M., 9 A.M., and 10 P.M., the blood samples being taken just before the saliva specimen. Breakfast

[1] Gerontologic Unit, The Allan Memorial Institute, Department of Psychiatry, McGill University, Montreal, Canada. This study was supported by grants from the Federal-Provincial Mental Health Grant to the Allan Memorial Institute of Psychiatry. The authors are grateful to Dr. R. Castro for his cooperation in finding suitable patients for study, and to Mrs. H. Berger and Messrs. L. Caplan, J. Berenson and A. Palffy for their technical assistance.

TABLE 1

The Sex Distribution of Patients with a Definite Diagnosis

	Males	Females
Endogenous depression	3	1
Involutional depression	3	0
Schizophrenia	7	3
Neurotic reaction	2	4
Alcoholism	1	0
Total.....	16	8

TABLE 2

The Sex Distribution of Patients with an Indefinite Diagnosis

	Male	Female
Schizophrenic or depressive reaction	2	3
Schizophrenic or neurotic reaction	2	3
Depressive or neurotic reaction	1	2
Neurotic reaction or personality disorder	2	1
Total.....	7	9

was taken at 8 A.M. The number of eosinophils per cubic millimeter of blood (absolute eosinophil count) was converted into a relative eosinophil value by expressing the absolute count of each person as a percentage of the mean of the counts for that person. The same procedure was followed in converting the salivary Na/K ratios (absolute Na/K values) into relative Na/K values.

Correlation, chi-square and *t*-tests were carried out on the data.

Psychiatrically, the patients were suffering from functional psychoses or neurotic reactions. Organic psychoses were excluded from this preliminary study. But even aside from this, the material was by no means unselected. The first observations suggested that there might be a difference in the diurnal pattern of the salivary Na/K ratio between cases in whom the clinical picture was stabilized and a nosological diagnosis was well established, and cases in whom the clinical picture was still in flux and the exact diagnosis not firmly established. Therefore, patients with functional psychoses or neurotic conditions were selected so as to fall in one of two groups. Group I comprised 24 cases in whom the nosological diagnosis could be considered as definitely established on the basis of their history, clinical symptomatology and response to previous treatment. Group II comprised 16 cases where according to the same criteria, a definite diagnosis could not be established during a sufficiently long observation period preceding the tests. The diagnostic distribution of the material is given in Tables 1 and 2. In view of the importance of the problem, special safeguards were used regarding the diagnosis. The first lead was taken from the notes of the attending psychiatrist and the staff in charge of the case. This was then checked independently by each of the two psychiatrists participating in this study and after careful consideration the individual case was then placed in one of the two groups before the laboratory tests were done. It should be mentioned that the patients with an indefinite diagnosis were not less disturbed than those with a definite diagnosis, and occasionally the former were even more so. Only, at the time of the study, their symptoms did not appear to organize themselves into any clear cut syndrome of a specific functional disorder. The biologist and the laboratory technicians in charge of the laboratory tests were completely unaware as to which group the individual patient belonged. Moreover, the technicians performing the sodium and potassium determination in the saliva did not know the results of the eosinophil counts.

In assessing whether or not the patients showed anxiety on the day of the test, use was also made of the notes of the nurse in charge. Moreover, at the time the nurse's notes were written neither the nurse in question nor the investigators on this project knew that the notes would be used in the project. Interest in this question arose only at the time the report was being prepared for publication.

RESULTS

Diurnal Variation of Eosinophils and Salivary Na/K. In the eosinophil counts, 27 of 35 patients (nine of 13 females and 18 of 22 males) showed the same pattern in which the 9 A.M. value was below both the 7 A.M. and 10 P.M. values. In the salivary Na/K, 23 out of 39 patients (nine of 16 females and 14 of 23 males) showed the same diurnal variation rhythm. The frequency of the occurrence of this pattern was highly significant among the males alone or females alone or males and females when taken together ($P < 0.001$).[2]

The eosinophil count per cu. mm. was 239 in 22 males and 233 in 13 females, a non-significant difference ($0.90 > P > 0.80$). This is in agreement with earlier studies (15). The salivary Na/K in males was 0.64 in 23 males and 0.52 in 16 females, also a non-significant finding ($0.20 > P > 0.10$). Earlier studies showed that there was no significant difference in the salivary Na/K between men and women under 40 (6). Most of our subjects were under 40.

Statistical analysis revealed that there was a significant positive correlation between the absolute eosinophil counts and the absolute Na/K ratios in males alone and in males and females together but not in females alone (Table 3). However, there was a significant positive eosinophil-Na/K correlation when expressed in relative values. This was true of the males alone, females alone, and in males and females together (Table 3). In these calculations the data of the 7 A.M., 9 A.M., and 10 P.M. were taken as one group. However, correlation coefficients of all the subjects were also calculated at each of the three time intervals. The *r* value was +0.3157 ($0.10 > P > 0.05$) at 7 A.M., +0.3770 ($0.05 > P > 0.02$) at 9 A.M. and + 0.5967 ($P < 0.001$) at 10 P.M.

[2] The determinations made at 7 A.M., 9 A.M., and 10 P.M. represent three points which when combined give a series of nine different curves. Therefore, the probability of obtaining any one curve is 1/9. For the salivary Na/K ratio, 39 subjects were tested and therefore the expected value was 39/9, while the value actually observed for one of the nine possible curves was 23. A chi-square test was conducted and a probability of $P < 0.001$ was obtained. The same calculation was done for the circulating eosinophil count and a similar P value obtained.

TABLE 3

The Significance of the Correlation Coefficients ("r" Values) Between the Circulating Eosinophil Counts and the Salivary NA/K in Psychiatric Patients

Sex	Absolute Data		Relative Data	
	r	P	r	P
Males	+0.4389 (22)	P<0.001	+0.3686 (22)	0.01>P>0.001
Females	+0.0834 (12)	P>0.10	+0.6403 (12)	P<0.001
Males and Females	+0.3253 (34)	P<0.001	+0.4883 (34)	P<0.001

The number in the parentheses gives the number of patients for which the adjacent "r" is calculated.

TABLE 4

Number of Patients With and Without Anxiety Having Normal and Abnormal Diurnal Rhythms of the Salivary NA/K

Group	Anxiety	Normal Rhythm	Abnormal Rhythm
I	With	5	5
	Without	14	0
	Total...	19	5
II	With	2	3
	Without	2	8
	Total...	4	11

Relation Between Psychiatric Diagnosis and Diurnal Variation of Eosinophil and Salivary Na/K. The data presented in Table 4 show that there were significantly more subjects in Group I with normal diurnal salivary Na/K rhythms than in Group II when all the subjects of each group are considered ($0.01 > P > 0.001$). This difference was even more marked if patients with manifest anxiety at the time of the test were excluded ($P < 0.001$).

Although there was no significant differ-

TABLE 5

Number of Patients With and Without Anxiety Having Normal and Abnormal Diurnal Rhythms of the Circulating Eosinophil Count

Group	Anxiety	Normal Rhythm	Abnormal Rhythm
I	With	6	1
	Without	11	2
	Total...	17	3
II	With	3	3
	Without	7	2
	Total..	10	5

ence between the number of patients with manifest anxiety at the time of the test in Group I and Group II ($0.90 > P > 0.80$, Table 4), significantly more of the patients with manifest anxiety in Group I showed disruptions of the normal diurnal variation pattern than did patients without manifest anxiety in the same group ($0.02 > P > 0.01$). Such a significant difference was not observed in the patients with and without manifest anxiety in Group II ($0.90 > P > 0.80$).

When the same comparisons were made in the case of the diurnal rhythm of the circulating eosinophil count as were done in the salivary Na/K, no significant differences were found to occur in any of the comparisons made ($P > 0.70$, Table 5).

Also investigated was the question whether there was a significant relationship between the patterns of the above diurnal variations and the EEG findings or the sedation threshold described by Shagass (16). No relationship could be found.

DISCUSSION

That there is a diurnal variation in the level of the salivary Na/K in humans has been unequivocally demonstrated as follows: salivary Na/K ratios were studied in four subjects over a minimum period of 17 consecutive days with samples being taken for study before breakfast, before lunch, before supper and before retiring. The pattern in which the "before lunch" sample was lower than the "before breakfast" sample was obtained 17 out of 17 days in the first subject, 17 out of 19 days in the second, 19 out of 21 in the third, and 22 out of 26 days in the fourth. (*cf.* 8, p. 714, Fig. 5) In another study (*cf.* 6, pp. 278–79, Table 4), 59 out of 62 subjects between the ages of 17 and 67 years (the age range of the present study) had a salivary Na/K ratio in which the "before lunch" sample was lower than the "before breakfast" sample. The actual magnitude of the difference between the "before breakfast" and "before lunch" varied from day to day, but it was so consistently in the same direction that there was no doubt of the existence of a pattern. Inasmuch as the great majority of normal persons have such a pattern, it was called "normal."

An intensive study was also made on the absolute venous blood eosinophil levels in 17 normal males at 16 different time intervals within a 24-hour period and in every one of them there was a drop in the count between 6:30 A.M. and 9:30 A.M. (*cf.* 11, p. 315, Table IV). Here again the magnitude of the difference varied from day to day but its great consistency left no doubt as regards the existence of a pattern. When the same studies were made in eight subjects with Addison's disease, bilateral adrenalectomy or hypopituitarism in none of the eight was there a fall in the circulating eosinophil count between 6:30 A.M. and 9:30 A.M. pointing to the role of the adrenal cortex in this phenomenon. Mice have also been shown to have a diurnal eosinophil rhythm. However, as they are nocturnal animals the low point is at night instead of in the morning (10). The importance of sugar-active adrenal cortical hormones in controlling the level of the circulating eosinophils has been demonstrated in numerous studies, and indeed, a bio-assay sensitive to small amounts of these hormones has been described (9, 17, 18).

The level of the salivary Na/K has also

been shown to be dependent on the adrenal cortex. Thus, ACTH administration has been shown to decrease the salivary Na/K in normal subjects (3, 5, 7, 8, 19, 20), but not in patients with Addison's disease (5). Desoxycorticosterone has been shown to decrease the salivary Na/K of normal subjects (5), and in patients with Addison's disease (4). Also, the Na/K of the saliva was higher in patients with Addison's disease and lower in patients with Cushing's disease than in normal subjects (5). Hence there appears to be an inverse relationship between the level of adrenal cortical function and the level of the salivary Na/K. Finally, a significant number of normal persons has been shown to have a definite diurnal pattern of the unstimulated salivary Na/K with a peak in early morning before arising, followed by a sharp drop later in the day until noon with a gradual rise thereafter (6). From this, it follows that the salt-regulating function of the adrenal cortex should be at a low point before breakfast, reach a high point before lunch, and decline gradually toward evening. This is the same rhythm that was revealed by the blood eosinophils as just described, and has also been demonstrated by the amounts of 17-ketosteroids and neutral reducing lipids excreted into the urine (14).

Inasmuch as the diurnal rhythm of both the blood eosinophils and the salivary Na/K appear to depend on the functional activity of the adrenal cortex, it would be expected that when taken simultaneously in the same patients, that there would be a significant positive correlation between the eosinophils and the Na/K. This was shown to be so except with regard to the absolute data of females only. However, as adrenal cortical function controls the *change* in the level of these functions, and not their absolute values, it was to be expected that expression of these data in relative terms would result in a higher degree of correlation between the circulating eosinophil counts and the salivary Na/K than was the case with the absolute data which varied considerably from patient to patient.

From the psychiatric point of view, several facts appear interesting. Out of 35 patients in whom the eosinophil level was determined, 27 had a normal pattern of diurnal variation. Out of 39 in whom the Na/K ratio was determined, 23 showed the normal diurnal variation. Finally, out of 34 patients in whom both determinations could be made, 17 had a normal pattern of diurnal variation and 17 did not. In other words, at least 50 per cent of the patients showed normal diurnal adrenal cortical variation when both tests were employed and about two-thirds had a normal rhythm when only one of the tests was used. This might appear the more remarkable as our cases were mostly "acute" in the sense that an acute psychosis or an acute exacerbation of a chronic psychosis necessitated admission to hospital. It has been reported that acute functional psychoses lead to detectable changes in adrenal cortical function (12, 13). This, however, is not in contradiction to our findings for our tests indicate the nature of the changes occurring in adrenal cortical function throughout the day, not the level at which the endocrine is functioning. For example, it has been demonstrated that when ACTH is administered, the adrenal cortex functions more actively but with the same diurnal rhythm as in its normal less active state (8).

The results are more interesting from the psychiatric point of view when the findings of Groups I and II are compared. In the salivary Na/K, 19 out of 24 patients had the normal diurnal variation pattern in Group I but only four out of 15 in Group II. Less clear was this difference in the diurnal variations of the eosinophil count, 17 out of 20 patients following the normal pattern in Group I, and ten out of 15 in Group II.

Our data revealed that patients in whom the psychiatric diagnosis could be determined with certainty showed a greater tend-

ency to have a normal diurnal rhythm of adrenal cortical activity than those in whom a diagnosis could not yet be made with certainty. It would seem therefore that the acute stress of a functional psychosis leads to discernible deviations of the normal adrenal cortical rhythm particularly in those cases where the clinical symptomatology does not permit a definite diagnosis. In other words, in cases where the clinical symptomatology is still so much in flux that a definite diagnosis can not be reached, the normal diurnal variation of the adrenal cortex seems to be more disturbed than in those cases where the clinical symptomatology has already reached such a degree of stability that a definite diagnosis can be established. One would expect that in the more stabilized cases (Group I) an additional stress would easier lead to manifest deviation from the norm of adrenal cortical function than in the cases still in flux (Group II). This is to be expected because a stress would tend to distort the normal diurnal rhythm, and in Group II over 70 per cent of patients already show such distortions as against 21 per cent in Group I. This expectation was borne out by the finding that anxiety at the time of the test acting as a stress, distorted the normal salivary Na/K pattern significantly more in patients of group I than in those of Group II.

In all this, the salt-activity (as revealed by the diurnal rhythm of salivary Na/K) rather than the sugar-active function of the adrenal cortex (as revealed by the diurnal rhythm of the circulating eosinophils) appeared to be the function in which differences between Group I and Group II were seen. This is of interest in view of the findings that the salt-active DCA does *not* eliminate mental symptoms in adrenal cortical insufficiency, while the sugar-active cortisone is more effective in such cases (1, 2).

SUMMARY

Unstimulated mixed saliva and venous blood were collected from 40 psychiatric patients at 7 A.M., 9 A.M. and 10 P.M. of the same day. Salivary sodium and potassium concentrations were determined by a flame photometric technique and circulating eosinophils were counted in the Fuchs-Rosenthal hemocytometer.

Earlier studies showed that a significant number of normal persons had diurnal patterns in the salivary Na/K and circulating eosinophil counts in which the 9 A.M. values are lower than both the 7 A.M. and 10 P.M. ones. The present study revealed that a significant number of psychiatric patients also had these normal diurnal patterns. Moreover, a significantly larger proportion of patients with a definitely diagnosed psychiatric illness of the functional type (Group I) had normal diurnal salivary Na/K patterns than did patients who also suffered from functional psychoses but in whom the nosological entity could not be established with certainty (Group II). Such differences between the two groups were not observed in the circulating eosinophil counts.

There were no significant differences between the relative proportion of male and female patients showing normal diurnal variation patterns in the salivary Na/K. This was also true of the circulating eosinophil counts. Neither were there any differences between the male and female patients in the absolute salivary Na/K levels and in the eosinophil counts.

Finally, there were significant positive correlations between the relative values of the salivary Na/K levels and the eosinophil counts of male and female patients considered separately or together.

REFERENCES

1. Cleghorn, R. A. Adrenal cortical insufficiency: psychological and neurological observations. Canad. M. A. J., **65**: 449–454, 1951.
2. Cleghorn, R. A. Steroid hormones in relation to neuropsychiatric disorders. In *Hormones*,

Brain Function, and Behavior, Hoagland, H., ed., pp. 3–19, Academic Press, New York, 1957.

3. Dreizen, S., Niedermeier, W., Reed, A. J. and Spies, T. M. The effect of ACTH and cortisone on the sodium and potassium levels of human saliva. J. Dent. Res., **31**: 271–280, 1952.
4. Frawley, T. F. and Forsham, P. H. The salivary Na/K ratio and adrenal salt regulating factors: prolonged salt retention with desoxycorticosterone trimethyl acetate. J. Clin. Endocrinol., **11**: 772–773, 1951.
5. Frawley, T. F. and Thorn, G. W. *Proc. Second Clin. ACTH Conf.*, Vol. 1 (Research), pp. 115–122. Blakiston Co., New York, 1951.
6. Grad, B. Diurnal, age and sex changes in the sodium and potassium concentration of human saliva. J. Gerontol., **9**: 276–286, 1954.
7. Grad, B. The influence of ACTH on the sodium and potassium content of human mixed saliva. Fed. Proc., **10**: 53, 1951.
8. Grad, B. The influence of ACTH on the sodium and potassium concentration of human mixed saliva. J. Clin. Endocrinol., **12**: 708–718, 1952.
9. Grad, B., Sher, O. and Symchowicz, S. Relative biological activity of some adrenal cortical steroids as determined by eosinopenic test. Proc. Soc. Exper. Biol. & Med., **84**: 1–5, 1953.
10. Halberg, F. and Visscher, M. B. Regular diurnal physiological variation in eosinophil levels in five stocks of mice. Proc. Soc. Exper. Biol. & Med., **75**: 846–847, 1950.
11. Halberg, F., Visscher, M. B., Flink, E. B., Berge, K. and Bock, F. Diurnal rhythmic changes in blood eosinophil levels in health and in certain diseases. Journal-Lancet, **71**: 312–319, 1951.
12. Pincus, G. and Hoagland, H. Adrenal cortical responses to stress in normal men and in those with personality disorders. Am. J. Psychiat., **106**: 641–650, 1950.
13. Pincus, G., Hoagland, H., Freeman, H., Elmadjian, F. and Romanoff, L. P. A study of pituitary-adrenocortical function in normal and psychotic men. Psychosom. Med., **11**: 74–101, 1949.
14. Pincus, G., Romanoff, L. P. and Carlo, J. A diurnal rhythm in the excretion of neutral reducing lipids by man and its relation to the 17-ketosteroid rhythm. J. Clin. Endocrinol., **8**: 221–226, 1948.
15. Rud, F. *The Eosinophil Count in Health and Mental Disease, a Biometrical Study*, pp. 240–244. Johan Grundt Tanum Vorlag, Oslo, 1947.
16. Shagass, C. The sedation threshold: a method for estimating tension in psychiatric patients. Electroencephalog. & Clin. Neurophysiol., **6**: 221–233, 1954.
17. Speirs, R. S. and Meyer, R. K. The effects of stress, adrenal and adrenocorticotrophic hormones on the circulating eosinophils of mice. Endocrinology, **45**: 403–429, 1949.
18. Speirs, R. S. and Meyer, R. K. A method of assaying adrenal cortical hormones bases on a decrease in the circulating eosinophil cells of adrenalectomized mice. Endocrinology, **48**: 316–326, 1951.
19. Thorn, G. W. Further clinical studies with ACTH and adrenal cortical hormones. In *Tr. 2nd Conf. on the Adrenal Cortex*, pp. 164–209. Josiah Macy Jr. Foundation, New York, 1951.
20. Warming-Larsen, A., Hamburger, C. and Sprechler, M. The influence of ACTH on the sodium and potassium concentration of human saliva. Acta endocrinol., **11**: 400–412, 1952.

PSYCHOLOGICAL TEST REPORTING: A PROBLEM IN COMMUNICATION BETWEEN PSYCHOLOGISTS AND PSYCHIATRISTS[1]

Introduction: The Scope of the Problem

LEOPOLD BELLAK, M.D.[2]

The papers that follow stem from a re-evaluation of the common observation that psychological testing plays a very important part in everyday psychopathological work. It constitutes the main area of contact between psychiatrist and clinical psychologist. Psychological testing is fraught with all the problems inherent in communication, those of interdisciplinary relationship more specifically, and some uniquely inherent in the subject matter.

The topic resolves itself quite naturally into certain areas.

1) There is the need for the psychiatrist to know enough about the psychological instrument to be able to understand test reporting for what it is—its assets and limitations.

The psychologist not only needs to know his tool and what kinds of data it can yield, he has to be able to formulate his findings in the most useful way. Amazingly little systematic attention has been paid to the writing of the psychological report.

2) Here then lies another large area of interprofessional concern: what is the best form in which results of psychological examination can be communicated? What material can the psychologist provide? What kinds of information does the psychiatrist need? Questions like these often make for lively discussions, and there is a pressing need for clarification.

3) In the clinical situation there is the ever-present question as to how much pre-testing information the psychological examiner should have. Some psychiatrists feel that if the psychologist has all available clinical information prior to examination, this may well contribute to a prejudgment of his results, may, in a word, make for bias at one level or another. On the other hand the psychologist may maintain that he cannot provide meaningful clinical data without some acquaintance with reality factors. It is of course clear that "blind" testing as a research stratagem is an entirely different matter from the psychological testing that goes on as a part of the total workaday clinical armamentarium.

4) That test data are derived from one level of behavior and the psychiatric interview from another level, is a fact frequently overlooked and not systematically enough taken into account. Helen Sargent has probably developed most clearly the conceptual hierarchy involved in diagnostic formulations; but the *integration of data from different levels of inference*, such as represented by the work of the psychologist and psychiatrist, need more extensive discussion and formulation.

5) The problems of communicating the data derived from psychological testing are variously inherent in the tools, in the concepts and in the organization of the report itself. Aside from the mechanics of the relationship, psychologists and psychiatrists are, of course, people. Their own emotions,

[1] A symposium held at the Annual Meeting of the American Psychiatric Association, San Francisco, May, 1958, under the joint sponsorship of the American Psychiatric Association and the Society for Projective Techniques, Inc. Dr. Leopold Bellak acted as chairman and Dr. Bruno Klopfer as discussant.

[2] Department of Psychiatry, City Hospital, Elmhurst, New York City, N. Y.

value systems, apperceptive distortions, and their semantic problems enter into this area of communication—as do such matters enter into all forms of relationship.

Psychological Test Report: A Communication Between Psychologist and Psychiatrist

HERMAN FEIFEL, PH.D.[a]

The work of the psychological tester is not truly completed until his significant findings have been communicated to others. In this sense, the test report can be viewed as a kind of bridge between individuals working on the same case. We have heard that to transmit meaningful information the test report must minimize jargon, reduce semantic obfuscations, and stress uniqueness rather than universals.

I should like to focus on another consequential aspect in this transmission process—the *frame of reference* from which the communication proceeds. Specifically, I refer to 1) the theoretical bases underlying psychological tests and 2) the reflection in test reports of changing concepts of personality. Both factors manifestly are interrelated and mutually influencing.

The propositions underlying testing are important to spell out because converging studies indicate that many semantic obstacles concerning clinical concepts between psychiatrist and psychologist stem from theoretical confusion. There are psychologists as well who are critical of psychological testing, describing it as "an empirical art that suffers from the lack of a solid theoretical foundation" (4). Nevertheless, psychologists do maintain certain working assumptions concerning the nature of tests and I propose briefly to examine the more obvious ones. (People like David Rapaport, David Wechsler, and Roy Schafer deserve commendation for their yeoman service in this regard.)

1. Psychological tests are a means of obtaining behavior samples, usually via ideational and motor activities, of the individual.

2. A person's characteristic style of thinking represents or reflects his constant adjustment efforts. (This operates in the realm of emotion and action as well.) His responses to various test items are the verbalized or motor end-products of thought processes touched off by those items; and definite causal ties exist between these end reactions and the person's overall ego organizing principles, *i.e.*, the typical or idiosyncratic ways by which each one of us comes to terms with the world, including the inner domain of feelings and impulses—the means we use of selecting and organizing internal and external stimuli. We should keep in mind that the test response itself is not a score which is really an abstraction derived from test responses. Scores are "designed to facilitate intra-and-inter-individual comparisons and, as such, are extremely useful in clinical testing," but "to reason only in terms of the score, or even score patterns, is to do violence to the nature of the raw material"—the test responses themselves (3). Fortunately, more and more psychologists are no longer being seduced by the correctness or incorrectness of the test response alone but are scrutinizing and evaluating the quality of the response, *i.e.*, the process or "how" and "why" of performance. Clinical psychology abounds in instances where results are obtained by un-

[a] Veterans Administration Mental Hygiene Clinic, Los Angeles, California.

expected roundabout means or where nonpassing scores conceal existing ability. It is obvious that studies and reports which try to appraise intellectual and emotional functioning and change by equating all successful responses, or failed ones for that matter, as though they had a single meaning, often confuse the periphery for the core of performance.

3. The configurational pattern or context in which a test response is embedded is another significant dimension for illumined interpretation. We have learned that similar formal test patterns can have dissimilar meanings in varying contexts. For example, on the Rorschach, a reduction of one M (a percept of human movement) on retesting may mean one thing when the drop is from one M to none at all; another, when it is from four Ms to three. And we have all seen that what appears as a pathological trend in one identity configuration turns out to be an asset in another one.

Recent findings have also enlarged the considerations of psychologists about the importance of the *social situational* context in which the testing itself takes place. The set, expectations, and feelings of both the tester and his subject can act as forceful influences on the test responses themselves. In similar vein, the psychological test report may represent not only the subject's productions, but the tester's *interpretation* of them as well. This realization is stimulating increasing study of the process of test interpretation, clinical judgment, and validation of the clinician.

4. Test interpretations and the diagnostic conclusions reached from them can be two relatively distinct parts of test analysis, though the latter are quite dependent on the former. An interpretation is a prediction about certain phenomena of behavior or thinking concerning a subject; a diagnostic conclusion generally subscribes to a nosological scheme. It is in this field that clinical psychologists often disagree among themselves or with psychiatrists by employing different diagnostic criteria (3).

5. To secure broad coverage of the various dimensions of ego functioning, a battery of tests should be given. This results in the subject's responding to a variety of problem situations. Because of the increased and different types of behavior samples thereby obtained, *e.g.*, requiring a specific answer to an arithmetic problem of this sort on the Wechsler-Bellevue, "8 men can finish a job in 6 days. How many men will be needed to finish it in a half-day?"; or asking the subject to make up a story to a picture card showing an elderly lady and young man on the TAT, we are in a position to tap various levels of psychic functioning and augment our ability to arrive at a rounded and hierarchical picture of the subject's functioning. The battery approach is also important in light of our knowledge that psychological maladjustment may encroach on certain test functions and responses and leave others relatively unimpaired. Additionally, it can help spotlight the *conditions* under which certain motives come into play, as well as their interrelationships.

6. Most clinical psychologists work from the conviction that adequate understanding of human behavior evolves only from studying the unsegmented person. Personality is viewed as a whole and not a random mixture of isolated traits. This does not mean that we cannot talk about discrete functions in our test reports if we realize that what we call functions, *i.e.*, concept formation, memory, etc. are merely different ways of looking at psychological functioning, and that psychological functioning is just one of the ways of looking at organismic functioning (2).

7. Finally, the widespread utilization of tests by psychiatry and psychology is predicated on the proposition that because of their indirect approach and relatively standardized nature they can secure material which is sometimes difficult to elicit and not readily apparent otherwise.

The assumptions just enumerated can be better grasped, I think, if we take a quick look at the major shift in personality theory during the past few decades. An inheritance from the 19th century was the concept of a lawful universe consisting of a great network of single, variable, causal relationships. Medicine was chiefly dominated by the successes emanating from the experimental laboratory where it seemed that the great mysteries of man's nature were being unraveled one by one. Germ orientation and the diseased organ were the kingpins of attention. The functioning organism was comprehended mainly within biophysical and chemical frames of reference. There was also general acceptance of evolutionary principles. Personality traits were considered as ingrained attributes of the individual and could be measured on objectively defined scales. Repercussions were evident in the testing movement where conspicuous attempts were launched to measure definitive specific traits and segments of behavior. Tests, for example, were created solely to measure ascendancy-submission or introversion-extraversion. In line with this guiding outlook, testing developed a decided experimental-statistical approach with stress on matters like sampling theory, test construction, standardization, and validation. The approach was overwhelmingly empirical and functional rather than theory-oriented. The Binet-Simon and Stanford-Binet came into being because "Binet was asked to predict scholastic achievement, not construct a theory or a test of intelligence as such. Rorschach did not set himself the task of constructing either a test or a general theory of personality. He was interested primarily in differential diagnosis of the mentally ill" (5). Psychometric and projective tests were generally empirical in design. The reason is not hard to find. The testing movement had its origin and owed its continued support to the needs and demands of various *service* agencies. And, frankly, things haven't changed much in this respect today.

Under the impetus of increasing knowledge in the fields of experimental neurology and the functioning of the ductless glands, and with the advent of psychoanalysis, the segmented-and-organ type of perception of human behavior began to give way. Experimental neurology indicated interrelationships of various parts of the body with central governing mechanisms of the nervous system; investigators like Pavlov, Cannon, and Selye highpointed the responsiveness of glandular activities to purposes and needs of the individual via delicate connections with the cerebrum and autonomic nervous system; and Freud's illuminating ideas helped open the door to understanding the vital links between mental life and somatic sources, and the tie-in between disturbances in organ functioning and unconscious mental processes. Disease and psychopathology were no longer what happened to a person's organs but, rather, to the whole person and were profoundly dependent upon the nature and fortunes of the individual's developmental process.

Allied concepts were also being expressed in psychology. A revolt took place against the concentration upon problems of sensation and perception and the reduction of consciousness to a form of atomistic sensationism. The Gestaltists set emphasis on the whole being more than the sum of its parts; Kurt Lewin announced his field theory concepts; Edward Tolman accented the importance of molar and goal-directed behavior. The organism was no longer an encapsulated unit but a node and organizing point in a field—a kind of open system of reciprocating outgoing and incoming energies. Thinking veered from fixed traits to interacting fields and transactional systems in which abilities, motives, and pathologies were vitally interrelated (7).

How did this mirror itself in the testing movement? Psychologists pay attention today, for example, not only to the I.Q., the level of intelligence, but also to its functioning under specific conditions. The efficiency

of intellectual functioning is related to the dynamics of the individual's adjustment as well as to specific illness and senescent change. Or in scrutinizing a subject's problem-solving capacity on a finger maze test or the Block Design subtest on the Wechsler-Bellevue, the tester considers such allied factors as the subject's frustration tolerance, his control over anxiety, level of aspiration, the possibility that the task may mobilize some childhood trauma, etc. (7). The tester no longer perceives his diagnostic task as solely arriving at a classificatory diagnosis but rather at contributing to the overall picture of what is happening. Hierarchical ordering and integration have become crucial problems. The tester is also not worried as much as he used to be about the low reliability of some of his tests because he senses that this may evidence the flexibility or versatility of the subject's varying behavior. He comprehends more clearly that what we say about a person's problem-solving ability or his aggressive potentialities becomes a statement about them under a *specific* set of circumstances. We can make contingent predictions from *specified* conditions. The glossing over of this point explains the failure of a lot of really good clinical studies to stand up on cross-validation. I think that when the relation of the subject to the detailed structure of the situation to be met is fully described, the situation can provide significant prediction above chance. Naturally, the gap between the testing situation and "real" life can never be wholly bridged. You can predict certain things, not *all* things.

A curious fact characterizes the general shift from statistical to psychological thinking concerning tests. What we behold in progress is not so much the creation of new tests as the trend to broader and richer interpretation of the data gathered from old ones. In the hands of a Rapaport who applies theoretical leverage, the Wechsler-Bellevue becomes not only a test of intelligence but one of personality and psychopathology as well.

At this stage, we are no longer justified in saying as one of our most distinguished confrères did ten years ago, at least not without some qualification, that "psychological testing is a pragmatic procedure protected by a thin veneer of statistical respectability and hardly in touch with the psychological reality of the problems it tackles" (1). It must be admitted, nonetheless, that with all our experimentation and the array of tests available in our armamentarium we still don't have tests that are wholly relevant to our tasks. Silvan Tomkins (5) has implied that, in a certain sense, "today's tests can be viewed as poorly designed experiments because they answer no one question well." The validity test studies which have been carried out have contributed, in essence, "little to the general fabric of science." The power of much of our present-day test interpretation arises from the circumstance that many of us work in dynamic clinical settings and are consequently sensitized to a richer understanding of the test data. This is all right for a short run, not the long haul. True, we haven't been sitting on our hands. We have followed the same testing instrument via longitudinal studies; we have critically observed it as a consequence of changes in condition.

I believe, however, as Tomkins has so persuasively argued, that maximal adequacy and meaningfulness of measurement come into being as an outgrowth of underlying theory. If theory and assumptions are stated explicitly, one can devise the techniques most appropriate and relevant for testing them. Granted that a group of measuring instruments may be used to discover laws. The point is that in the field of personality our problem in general is that we don't quite know, except in adumbrative fashion, which aspects are significant and what the important relationships among them might be. Adequacy of measurement

is a function of the adequacy of underlying theory. In such sequence, a test can become an experiment designed to confirm or reject an hypothesis (5). In addition, I feel that a good portion of the semantic confusion between psychologist and psychiatrist originating from verbal discrepancies between them can be reconciled by deeper knowledge about the phenomena of human behavior. There are encouraging signs that we are moving in this general direction. The work of people like Rapaport and Schafer, among others, is slowly narrowing the gap between practice and theory.

Let us not deceive ourselves. The psychological tests we possess today—complementing the techniques of observation, the interview, genetic material, and the experimental laboratory—are cogent tools in extending our grasp of why people think and act the way they do, even though they may not provide us with as systematic or reliable data as we would prefer, or might gain from a well-designed experiment. As David Wechsler (6) once said, tests may not be as good as some of us claim or want them to be, but our claims are much better than we think.

REFERENCES

1. Rapaport, D. Discussion on the psychologist in the clinic setting, Round table, 1947, S. J. Beck, Chairman. Am. J. Orthopsychiat., **18**: 493–497, 1948.
2. Rapaport, D. Non-projective personality tests; principles underlying non-projective tests of personality. Ann. New York Acad. Sc., **46**: 643–652, 1946.
3. Schafer, R.: *The Clinical Application of Psychological Tests.* International Universities Press, New York, 1948.
4. Shaffer, L. F. Theoretical bases of psychometric tests. In *Relation of Psychological Tests to Psychiatry,* Hoch, P. H. and Zubin, J., eds., pp. 26–38. Grune & Stratton, New York, 1952.
5. Tomkins, S. S. A discussion of "personality structure and personality measurement" of R. B. Cattell. In *Invitational Conference on Testing Problems, 1951,* pp. 97–101. Educational Testing Service, Princeton, 1952.
6. Wechsler, D. Discussion, I. In *Relation of Psychological Tests to Psychiatry,* Hoch, P. H. and Zubin, J., eds., pp. 39–44. Grune & Stratton, New York, 1952.
7. White, R. W. What is tested by psychological tests? In *Relation of Psychological Tests to Psychiatry,* Hoch, P. H. and Zubin, J., eds., pp. 3–14. Grune & Stratton, New York, 1952.

Different Levels of Sampling Behavior in the Diagnostic Psychiatric Interview and the Test Situation

BELA MITTELMANN, M.D.[1]

In general one may say that the projective psychological techniques probe deeper personality levels (along with the superficial ones) than the initial psychiatric interview or interviews. Some reservations must be made, of course. (One occasionally sees patients with overt schizophrenia in which one or another of the projective tests has missed the diagnosis.) A critical question centers about the extent to which the projective findings affect the patient's actual treatment and the planning for the patient's treatment. The situations encountered may be divided into three broad categories. 1) The diagnosis based on the projective tests and the diagnosis based on the initial interview are in partial or total disagreement. 2) The projective tests suggest implications of symptoms or of conflicts and dynamics that are additional to what was found in the psychiatric interview. For example, the psychiatric interview may show a psychogenic depression and the Rorschach may also suggest the likelihood of severe anxiety. In such cases, the findings of the projective test are of essentially the same quality as those re-

[1] Department of Psychiatry, Albert Einstein College of Medicine, New York, N. Y.

vealed by the psychiatric interview, *e.g.* the patient has a psychoneurotic type of depression and the Rorschach or other projective tests would indicate that he also has a psychoneurotic type of anxiety. The results obtained from the two approaches, then, are *congruent* although not identical. 3) The projective tests give indications of psychological processes which are of a graver nature than those revealed by the initial psychiatric interview or interviews, *e.g.* the psychiatric interview indicates a psychoneurotic process and the projective methods suggest the presence of psychotic processes or at least a psychotic potential.

In general, my opinion is that the nature of the treatment should be determined by the psychiatric interview but the projective findings should be used as pointers and/or as alerting signals for various eventualities.

Let us apply this general statement to the three kinds of situations mentioned above. 1) If the diagnostic categories indicated by the interviews and test are different, the therapist may probe cautiously but more directly as to clinical manifestations possibly missed, and then base the treatment mainly on the resultant psychiatric picture. As treatment proceeds, it may develop that the initial test findings receive more support. The therapist should keep an open mind for such a possibility until the case is thoroughly understood.

2) Let me illustrate the idea of congruent findings with a brief clinical example. A college student came for consultation because of a psychoneurotic depressive picture. The Rorschach findings indicated strong anxiety with the assumption of likely overt manifestations. After being alerted to this possibility the therapist soon found out in the treatment that indeed the patient did have such manifestations. On and off, while lying in bed in a semi-awake state, the patient would feel that two arms were reaching out from under the bed to embrace her and crush her.

3) Test-obtained indications of a psychotic potential are occasionally not observed in initial interviews. In the course of psychiatric treatment it may happen that the suggestions of the projective material are validated rather quickly; on the other hand no supporting evidence may appear over a long period of treatment. The tests' indications may, of course, be wrong. Yet there are cases in which a genuine psychotic potential does not appear while the patient lives in a suitably supportive environment, manifesting itself only when conditions become unduly difficult. Again as an illustration, a 23 year old college student had been suffering from severe recurrent asthma from the age of five. In the psychiatric interview, it was easy to establish contact with her. She showed a mobile emotional responsiveness. The projective tests, particularly the Rorschach, indicated a psychotic potential. No such signs were clinically evident during the first year of treatment when the patient made excellent progress. Asthmatic symptoms cleared and she was able to engage in activities which she had never been able to enjoy in her past life, for example, dancing. She was able to assert herself against a rather difficult father. After nine months of psychotherapy the father announced to the patient that he would finance the treatment for another three months only. Concurrently with this development the patient was having problems in one of her courses in college, experimental psychology. In my experience this is often a particularly difficult course for emotionally disturbed patients, and in this instance the course was conducted by a particularly tough, sarcastic and demanding instructor. The patient's asthma returned at this point. She was not relieved by the assurance of her therapist that he would continue the treatment even if the father was reluctant to finance it beyond a year. The therapist further pointed out that the father might eventually be willing to cooperate. Efforts to make the father change his general behavior and attitude were of no avail. There was no progress in the psychothera-

peutic treatment. The first frank psychotic manifestation occurred when the patient telephoned the therapist shortly before the term examinations to say that she planned to announce to her family that masturbation at the age of five had ruined her mind. The therapist dissuaded her from doing this; however, acting as the proctor at one of the patient's term examinations was the instructor of experimental psychology whose course she had dropped. In the middle of this examination she suddenly got up and left, and soon after attempted suicide by taking an inadequate amount of phenobarbital. When the father learned of the suicidal attempt, his reported reaction was to ask the patient, "Do you know that I could have you arrested for attempting suicide?" Subsequently the patient became completely detached in psychiatric treatment and there was absolutely no progress. She then developed a generalized dermatitis and the family decided to refer her to a dermatologist. Psychotherapy was terminated. Leaving the hospital where she had been sent for dermatological care, the patient went to a library to learn the adequate amount of phenobarbital, secured it, and successfully committed suicide.

This is a case of a psychotic potential—initially suggested by results of projective psychological examination—emerging only after a considerable period of time, when the patient's general life situation became acutely stressful.

In summary, and with some reservations, it is my opinion that projective tests do indeed probe a deeper level of the psyche than initial psychiatric interview or interviews. In this connection I have suggested three kinds of clinical situations or categories, and have briefly illustrated them. Primary decisions about the management of the patient clearly should be based on the psychiatric interview. However, projective findings should certainly be taken into consideration as additional pointers and alerting or warning signals. A psychotic potential suggested by projective findings may not become clinically evident at all unless the patient's environmental situation becomes acutely stressful. This is an additional warning in the direction that in such instances, contact with the environment is an indispensable and critical part of the treatment process.

Psychological Test Reports: Universal or Discriminating

BERTRAM R. FORER, PH.D.[5]

A psychological test report contains a number of statements about a patient gleaned by a test expert from projective and other data. Regardless of the test battery from which the raw data come, the clinician makes statements which he thinks are true about the patient, statements which he *happened* to think of or be sensitive to. Tests do not automatically yield diagnostic statements or decide what is important.

Davenport (3), working with reports based upon Thematic Apperception Tests, found different emphases in different reports, but the differences proved to be more imagined than real. The differential occurrence of statements throughout the reports did not reflect the exclusive presence or absence of the characteristics in the patients. The most commonly used kinds of statements were judged to be applicable to almost every patient in the group. The clinicians in the study seemed to prefer descriptive or dynamic statements that were found to be almost universally applicable. These are, of course, the

[5] Veterans Administration Regional Office, Los Angeles, California.

very statements that have no validity in distinguishing one patient from another.

In a study by Datel and Gengerelli (2) clinicians matched Rorschach protocols and reports based upon them. Here the matchings were at their best when the patient group was very heterogeneous, hence minimum overlap in characteristics could be expected. Most of the reports that proved to be discriminating seemed useful for describing individuality in only a very broad way, *e.g.* between diagnostic groups, but contained few cues to differential validity. They could not discriminate between persons having the same diagnosis. To be sure, the matchings for groups were above chance, but not consistent enough to be of clinical worth.

A paper to be published by Blank (1) compares ratings by therapists and diagnostic testers on personality variables of patients in therapy. A rather curious finding is that blind ratings performed by psychologists who knew *only* that these were patients in therapy and had no access to tests or other clinical information, showed as much agreement with therapists' evaluations of the patients as did the ratings based upon Rorschach examinations. Incidentally, agreements of psychologist test raters with psychologist therapists were much higher than with psychiatrists or social workers.

Two implications of these findings are that: 1) many diagnostic reports are applicable to almost every patient, and 2) valid clinical inferences can be made with essentially "no" data.

Some time prior to the studies cited above, the writer presented individual personality descriptions to a group of college students (4). The descriptions were presumably based upon a group-administered interest test. Students rated their personal descriptions as a whole and each specific statement in the descriptions in terms of their accuracy as they saw themselves. The group almost unanimously accepted the descriptions of themselves as valid. They did *not* know that everyone had received the *same* personality description. Seventy-five per cent of the statements—which, incidentally, had been borrowed from an astrology book—were accepted as true by almost everyone in the group. These items were apparently universally valid and so practically useless.

It seems, then, that we have a problem in preparing psychological reports that involves the generality of the statements comprising the reports. What does it mean when we make a clinical statement that is universally valid? It is equivalent to saying at the physical level that a person has two arms. To be sure it is a valid statement. But, is it helpful diagnostically to know that a given patient has struggles with primitive impulses or an oedipal problem, employs repression, has not attained his ego-ideal, has latent potentialities for homosexual, aggressive or other strivings?

In psychometric parlance these items have no power of discrimination. They inflate the psychological report and lower its reliability. They contribute to lowered validity of the total report by reducing the number of truly discriminating features. More seriously, reports that contain an excessive number of universally valid statements tend to beguile the therapist into believing that he has obtained more information of diagnostic significance than he actually has. In some of the studies quoted above, the confidence of those who received the reports that they had received significant information was extremely high. It is because of their patent validity that universal statements foster unsubstantiated confidence that can be confused with understanding.

Regardless of the theory of personality to which one subscribes, diagnostic statements should fulfill several criteria. Some are as follows: 1) A statement should be true, that is, valid for the patient it purports to describes. 2) It should not be true of everyone. It should not, in fact, ordinarily be true of all patients. It should be differentially valid and enable persons to be classified into at

least two groups. 3) In a report, the separate statements should vary in the amount of differential occurrence. Some items should differentiate groups on a broad basis such as diagnosis, intellectual level and the like. Others should occur with less frequency or generality and serve to specify successively more detailed differences between persons having many other characteristics in common. 4) A report should contain statements which describe independent dimensions or characteristics so that no two individuals will be described with the same set of descriptive statements. The degree of elaboration of individual uniqueness will, of course, depend upon the diagnostic requirement at hand. At times a single statement may conceivably suffice for the purpose at hand. On other occasions a multiplicity of unique statements will be required.

The studies quoted previously—in addition to abundant other evidence in the research literature—suggest something about the nature of differentially valid or discriminating items. The first consideration is that virtually all traits, impulses and personality structures are universal in occurrence. And it is not in their presence or absence that people differ. Persons vary not so much in the presence or absence of given traits or wishes, such as aggression, anxiety, dependency or orality; as in the frequency, form intensity and social context of their expression. We might say that everyone manifests aggression in some way and in some situations. Individual A will show his aggressive urges in sarcasm, B in withdrawal or silence, C in physical assault and D in verbal argument.

A second important consideration is the threshold for any given form of behavior. One individual whose Rorschach protocol reveals a latent psychosis will manifest his psychotic underpinnings independently of the stimulating situation, another only in response to verbal abuse, another on the couch, another after two years of uncovering therapy, another only under the most stressful of circumstance, such as isolation from stimuli. We have some research data that describe significant differences among diagnostic groups both in the forms of aggression most commonly expressed and in the threshold for any given form of aggression. In short, the likelihood or probability of occurrence of a given action is as important an item of information as the nature of the act itself.

A third and most important factor in documenting the individual's uniqueness is the nature of the stimulus or situational context to which the act is a response. Just as a patient will react to different Rorschach blots or TAT cards with different content, affects or wish expressions, so he will react differentially to different social situations. Knowing the content of behavior without reference to the stimulus situation in which it developed provides only the most minimal cues for predicting how the patient will react to a given therapeutic or other social situation. Quite different clinical inferences will follow from our knowledge that his anger is aroused by the sight of a violin or a picture of a murder. In fact, a significant value of diagnostic testing is its use of standardized situations that are more structured than a therapeutic interview. The more we can specify the nature of the situation which evokes anger, fantasy or overt, toward whom, in response to what kind of interpersonal relationship, the more closely we approach the useful description of an individual. The more we shall have escaped the deceptive comfort of diagnostic stereotypes, however true they may be.

Again, these considerations do not necessarily imply a particular theory of personality. What they require is that the patient *not* be envisaged as a closed, autonomous system which emits behavior entirely on an endogenous basis. We suggest that a mapping of the ego or other enduring aspects of the person is not complete without specification of how this ego deals with a variety of concrete and specified data, motivational,

interpersonal and physical, the kinds of data which might be predictive of therapeutic response or describe areas of maladjustment.

This brings us to a final point, having to do with the varieties of behavior sampled by the tests. We need to face the issue of whether our projective tests provide evidence about *enough* and significant aspects of our patients' behavior. The day of the single universal test is happily past. New techniques are being developed that provide new stimulus situations and may enable us to predict other aspects of social and therapeutic response. While many of the new test stimuli seem to add nothing new, others have been designed to provide evidence on new dimensions in specific ways. They assume, sometimes only tacitly, that behavior occurs *only* with reference to stimuli and that control of the stimulus, projective or therapeutic, is a basic tool in our forcing the personality to yield its secrets.

REFERENCES

1. Blank, L. Suggestions for research with projective techniques. J. Proj. Tech., **22**: 263–266, 1958.
2. Datel, W. E. and Gengerelli, J. A. Reliability of Rorschach interpretations. J. Proj. Tech., **19**: 372–391, 1955.
3. Davenport, B. F. The semantic validity of TAT interpretations. J. Consult. Psychol., **16**: 171–175, 1952.
4. Forer, B. R. The fallacy of personal validation: a classroom demonstration of gullibility. J. Abnorm. & Social Psychol., **44**: 118–123, 1949.

The Psychological Report as a Problem in Interdisciplinary Communication

WALTER G. KLOPFER, Ph.D.[6]

The purpose of a psychological report is to communicate the results of a psychological evaluation to certain specified individuals or groups. The data are derived from behavior in a standardized situation. It is assumed that behavior is always determined and that it is basically consistent. The further assumption is made that, from the individual's behavior in this particular situation, it is possible to predict his behavior in situations not directly observed.

The purpose of a psychological report is *not* formal diagnostic classification, as this is ordinarily done by the interdisciplinary team on the basis of all the information available. The purpose of a psychological report is *not* to demonstrate the erudition of the psychologist nor to serve him as a platform for expounding his pet theories. The psychological report should *not* be the medium by which the psychologist demonstrates his professional competence to the referrent (as this competence should be assumed by virtue of the referral having been made). Rather, the clinical psychologist, like any other professional consultant, should use the report as a medium through which to describe his findings and give his impressions as clearly and concisely as possible. In view of the present stage of relative ignorance concerning the kinds of predictions which properly can be made from psychological findings, caution should certainly be exercised throughout.

Some psychological clinicians organize the summaries of their findings in terms of information sources. In effect, data are reported as having been elicited from particular psychological instruments or even from specific stimulus materials that are part of a diagnostic technique. This kind of organ-

[6] Department of Psychology, University of Portland, Portland, Oregon.

ization may occasionally take the place of individual supervision but is certainly not designed to communicate adequately in an interdisciplinary setting.

In order to be of the greatest value in the usual interdisciplinary setting, psychological reports should be organized according to personality areas such as intellectual aspects, affective aspects, interpersonal aspects, diagnostic implications, prognostic implications, etc. The particular organization is not as important as that the organization be comprehensible and comprehensive. Hypotheses derived from the psychological instruments should be delineated and separated from the impressions gained on the basis of other sources, as for example the clinical impression of the psychologist. Although the psychological report should clearly focus on the outstanding features of the case and should always deal with the questions specifically cited in the referral, they should not be limited in this sense but should include all relevant findings which may be of help in the overall understanding of the individual.

If the psychological report is to efficiently serve the purposes of interdisciplinary communication, language which is technical to the field of clinical psychology, referring for instance to particular ways of sorting test data, is to be assiduously avoided. Language which relies upon the utilization of a specific theory of personality, *e.g.* psychoanalytic theory, learning theory, can be employed as long as everyone professionally involved in the setting can accept and understand the terminology. If this is not the case, the use of such specific terminology may well serve as an unnecessary barrier to communication. It has been the experience of the writer that very simple and consensually validated language usually communicates best. If the psychological report is to be utilized for a variety of purposes and is addressed to semi-professional and non-professional referrents as well as professional ones, it may be necessary to prepare several different reports. A report designed to serve too many different purposes may serve none adequately.

One of the important problems in the communication of psychological findings has to do with the "level" at which a given interpretation is being made. Some of the predictions from psychological instruments have to do with material which is nonconscious in nature and which may have no recognizable behavioral manifestation. If this is the case, it should be made clear. Furthermore, the psychological evaluation should distinguish between material of which the patient is consciously aware, and that which may be quite evident to other people behaviorally but which the patient does not have under conscious control. It is obvious that a clear specification of levels in the psychological report will greatly facilitate the integration of psychological data with data from other sources. It is further felt that accuracy in this respect will help to emphasize the point that the main value of psychological evaluations is to shed new light on the understanding of the individual being discussed and not to agree or disagree with the findings derived from other media.

It is my opinion that some of the findings of the psychological examination can best be presented in oral form. This is the point at which illustrations can be given and interpretations can be documented. Wherever it is possible, interdisciplinary communication can be greatly facilitated by extensive verbal interaction between the clinical psychologist and his colleagues. The oral psychological report is considered the method of choice when communicating to school teachers, social workers, nurses and other individuals whose professional training includes psychological matters only tangentially. And when psychological findings are being presented in a staff meeting attended by representatives of various pro-

fessions, oral elaboration or abstraction of the results of psychological examination will surely facilitate interdisciplinary communication. In a real sense the sole and only legitimate purpose of the psychological report is that of communication.

The Psychiatrist's Decision to Refer a Patient for Psychological Testing

EUGENE B. BRODY, M.D.[7]

This has been called the age of anxiety; some people call it the era of tranquilization; it is certainly the time of the self-conscious man. This strong drive toward self-understanding has resulted in a rather unseemly prosperity of those who consider themselves professionals, in a field where there are so many amateurs. Further, it has resulted in an enormous proliferation of the "professionals." Where, formerly, a medicine man or soothsayer might have done the job himself, we now have psychiatrists, psychoanalysts, clinical psychologists, psychiatric social workers, psychiatric nurses, vocational counselors, lawyers and judges, group workers, and many other people with varying degrees of training, plus a multitude who, by combining an unprotected professional trade name with native talent, dispense their wisdom as freely as the rest. As one writer has put it, "there are many small gods abroad in the land."

This living together at close quarters may be one of the facts requiring some deliberate attention to the problem of interprofessional communication. It is almost sad, though, that our concern is with communication between psychiatrist and psychologist. We do have the problems that come when any two groups have increasingly overlapping functions. But we have more in common. Psychologists and psychiatrists are the only two groups trained professionally at the doctoral level, with persisting concern for research in behavior, who are also concerned with clinical practice. I suspect that the future may find us less concerned with communication with each other than with keeping our heads above water in a mounting tide of relatively untrained practitioners.

Now, what about the problems of communication as they relate to indications for psychological testing? This question recognizes that we are dealing with a complex social process involving two people—two people with differing training and differing professional identifications, who *both* conceive of themselves as members of helping professions and as investigators of behavior. The issue, then, is not why a patient should or should not receive psychological tests. It is why the psychiatrist does or does not refer his patient to a psychologist. This issue assumes particular significance in the light of Dr. William Menninger's statement in 1948, when he was President of the American Psychiatric Association. He said, "The diagnostic function of the clinical psychologist is now so well established . . . that the competent psychiatrist . . . would no more exclude the special techniques of the psychologist in his diagnostic studies than would a capable internist routinely exclude the findings of the roentgenologist" (3).

The working hypothesis central to this discussion is that the psychiatrist's request for psychological testing is not simply a dispassionate, objective effort to obtain scientific evidence which will be useful in his attempts to understand and treat his pa-

[7] Department of Psychiatry, University of Maryland School of Medicine, Baltimore, Maryland.

tient. It is a social process involving his attitude toward psychologists, toward psychological testing, his identification with his own profession, and his insecurities about his own status.

There are some apparent differences between the two professional participants in his social process (1). Clinical psychologists are relatively new on the scene, and their training encourages them to be skeptical and questioning. As a profession, clinical psychology is young, striving, and aggressive. Psychiatrists receive their basic training in a highly structured atmosphere, and they lean upon the tradition of medicine. Their training begins with the study of the cadaver; Fliess (2), in his discussion of the 'autopsic encumbrance,' considers the possibility that the doctor's early training may even impair his later functioning as a psychotherapist.

Both participants share the social role of the healer which is thrust upon any practitioner by virtue of the function he tries to perform. Internal conflict about this role may be reflected in feelings about professional colleagues of different disciplines who engage in similar activities. A psychologist's defense against the anxiety attached to this role may be reflected in overt or covert attacks upon the psychiatrist's ability or even right to perform functions he considers legitimately and historically his. Many psychiatrists respond by an unwillingness to have any professional contact with psychologists, except for an occasional tame psychometrician.

There are other factors which make for misunderstanding. The psychiatrist, a doctor concerned with what he sees as illness, may resent the many roles in advertising, industry, and government which the psychologist can assume at the same time that he functions in the medical field. Certainly the temptation is strong for the psychiatrist, forgetting the still uneven quality of his own specialty training, to emphasize the inadequacies of the psychologist's clinical training and experience. The feeling that the psychologist has not developed the traditional medical sense of responsibility for his patients or that he may regard the patient more as an object of research than as someone to be helped, may be used by the psychiatrist in his attempts to rationalize not only his failure to employ psychological testing, but his tendency to ignore, if not to be contemptuous of, the scientific contribution of psychology. This tendency may be clearly observed in psychiatric residents who mask their own insecurity by an uncritical acceptance of clinical precepts by senior men in their own field and by a tendency to regard most research by psychologists as sterile and far removed from the "realities" of life.

The context in which the psychiatrist operates is important. Do psychiatrists in private practice refer to psychologists as often or for the same reasons as those in clinic or hospital practice? In many public clinics, patients are routinely referred for psychological testing. This dependence not only upon the report of the psychologist, but upon the opinion of the social worker, may be a function of the insecurity, youth, or inadequate training of the psychiatrist in that setting. Economic factors are important here. There are more psychologists and social workers available for clinic service, where salaries, while good for them, cannot compete with the rewards of private psychiatric practice. The psychiatrist's time must be carefully husbanded. Typically the clinic patient is first interviewed by a social worker and examined by a psychologist. The psychiatrist, on the basis of the preliminary work of his two colleagues, may then arrive at a diagnostic or therapeutic conclusion after a relatively brief interview.

The complications which may arise from the fact that the psychiatrist is often less sophisticated and experienced than his social work colleague has been noted in the social work literature.

What are some of the hidden functions

of psychological test referral for the psychiatrist? The referral may be made in order to give the patient some feeling that his doctor's impressions have a sound scientific backing. "Tests" are often perceived as having a special value all their own in this technologically-oriented society. They are thought of as "objective" in contrast to the subjective opinions of the psychiatrist.

Again, negative instances are hard to use when it comes to integrating data about the patient. It would be important to learn how many psychiatrists ask for tests only when they have essentially negative information, and to learn how heavily they depend upon the tests to give them positive data. It is often impressive to note the joy with which people who ought to know better may leap upon a positive finding or two in a psychological report. One wonders at times, in reading psychological reports, if there might be a tendency on the part of psychologists to offer positive information as an unconscious way of justifying the testing procedure. More often than not the nature of the report which the doctor receives is a function of the way in which he asks for it.

Then there is the problem of getting consultative help. Psychological testing provides the clinician with a rapid feedback without risking some loss of status in his patient's eyes by asking for consultation from another physician.

A final example of the hidden functions of testing is the use of psychological referrals in the service of the psychiatrist's defensive needs with certain difficult patients. Here, the doctor may unconsciously wish to interpose the psychologist as a barrier between himself and his patient, or to use the apparently impersonal test as a substitute target so that he himself will not become the object of his patient's sexual or aggressive desires.

With these examples in mind, what are examples of the opposite? What are some of the hidden functions of the decision *not* to refer the patient for testing? Psychological testing may impose a considerable degree of delay upon the diagnostic and therapeutic process. The pay-off in medicine, as in other fields, is usually dependent upon the ability to use an immediately available cue, one which can be seen, heard, or smelled. This gives prestige, reinforces the patient's perceptions of the doctor's omnipotence, and permits him to act immediately. In this instance, the patient's attitudes and social class, as well as the nature of his symptoms, may determine the psychiatrist's decision.

Studies of the behavior of research groups have suggested that unconscious guilt and anxiety are more prominent in investigations of human behavior than in other fields (4). In the eyes of some, the use of psychological testing is a way of trapping the patient into giving information about himself, of which he is unaware.

Psychiatrists may avoid test referrals because they resent the omniscient report of the man who gives the "objective" tests; they know that the opinion of the psychologists is generally determined less by objective scoring than by his own clinical orientation and experience. If this is so, the psychiatrist may feel that he himself could arrive at conclusions which are just as, or even more, valid, on the basis of the test protocols or of the interview behavior of the patient.

Finally, the psychiatric interview and the history usually provide so much material in themselves that the process of integrating them is a real problem. It is very difficult to deal simultaneously with many pieces of information at once. One way to get around the problem is to restrict the number of information units with which one deals. The psychiatrist who is excessively dependent on psychological referral may restrict decision-making data to the information contained in a test report. On the other hand, tests may be avoided in the effort to restrict

the additional inflow of signs which require integration.

In summary, we are dealing here not with the established value of psychological tests as behavior-sampling devices. We are dealing with the generally unidentified factors which may result in the psychiatrist's depriving his patient of their benefit, or in his developing an unwarranted dependence upon them. Some of these factors are economic. There are also circumstances in which avoidance of psychological testing or unusual dependence upon tests is justified and rational. Many of the factors, however, are involved in the psychiatrist's perception of the person of the psychologist and of the nature of his tools. These influence the social process which is referral.

REFERENCES

1. Brody, E. B. Interprofessional relations or psychologists and psychiatrists are human too, only more so. Am. J. Psychol., **11**: 105–111, 1956.
2. Fliess, R. The autopsic encumbrance. Internat. J. Psycho-Analysis, **35**: 8–12, 1954.
3. Menninger, W. C. Psychiatry and psychology. Am. J. Psychiat., **105**: 389–390, 1948.
4. Redlich, F. C. and Brody, E. B. Emotional problems of interdisciplinary research in psychiatry. Psychiatry, **18**: 233–239, 1955.

BRIEF COMMUNICATION

THE AVAILABILITY OF HEXAFLUORODIETHYL ETHER BY INTRAVENOUS INJECTION AS A CONVULSANT IN PSYCHIATRIC TREATMENT

JOHN C. KRANTZ, JR., PH.D.,[1] L. LAVAN MANCHEY, PH.D.,[1] EDWARD B. TRUITT, JR., PH.D.,[1] ALFRED S. C. LING, M.S.[1] AND ALBERT A. KURLAND, M.D.[2]

Hexafluorodiethyl ether,

$$CF_3 \cdot CH_2—O—CH_2 \cdot CF_3,$$

(Indoklon®) was found to elicit convulsive seizures in laboratory animals when exposed to low concentrations of its vapors (6). This observation prompted its use as a convulsant in psychiatric treatment employing anesthetic technics for the inhalation of its vapors (3, 5). This procedure has been employed in more than 1500 treatments. It occurred to certain psychiatrists observing the administration of the drug that the treatment procedure would be simplified if the compound were available by vein. Our endeavors to achieve this end were prompted mainly by the urging of Dr. Lothar B. Kalinowski (4).

These studies are concerned with the preparation of a suitable solution of Indoklon for intravenous administration, its pharmacologic evaluation for safety, and the preliminary trials in man.

PREPARATION OF INDOKLON SOLUTION

Indoklon is a colorless, mobile liquid which emits a mild, pleasant ethereal odor. The liquid boils at 63.9° C. and has a specific gravity of 1.41 20°/4°. Indoklon is soluble in alcohol but soluble in water only to the extent of 0.5 ml./100 ml. The compound is extraordinarily stable and refractory to the metabolic processes of the animal body. Thus the problem presented itself of injecting a volatile substance immiscible with water and blood. A literature search indicated that this was achieved by Burkhardt (1) in the intravenous administration of ether and of chloroform dissolved in saline solution for the production of surgical anesthesia. Burkhardt's procedure proved impractical with Indoklon owing to its lesser solubility in water and the necessity for injection of large volumes of salt solution rapidly (200 to 300 ml.).

Accordingly, another solvent was sought which would permit increased concentrations of Indoklon and prove to be compatible with blood. After many unsuccessful trials with various solvents it appeared that polyethylene glycol 300 (Carbowax 300) was admirably suited for this purpose. The solvent is miscible in all proportions with Indoklon. When, however, water is added, separation of the two components occurs. The most satisfactory solution for injection was found to be that composed of five per cent Indoklon, 76 per cent Carbowax 300, and 19 per cent water, each computed on a volume basis. The solution withstands autoclaving at 15 pounds of pressure for 25 minutes for sterilization.

The toxicity of Carbowax 300 has been studied extensively by Smyth *et al.* (7) and Carpenter and Shaffer (2). It is not metabolized in the body and is excreted by the kidneys in urine within a 24-hour period after administration. The LD_{50}, injected

[1] Department of Pharmacology, University of Maryland School of Medicine, Baltimore, Maryland. This investigation was supported in part by Research Grant (M2540) from the National Institute of Mental Health, and by a grant from the Ohio Chemical & Surgical Equipment Co. (a division of Air Reduction Company, Inc.), New York, N. Y.

[2] Spring Grove State Hospital, Baltimore, Maryland.

rapidly intravenously undiluted into the rat, was found to be 7.1 ml./kg., which would correspond to approximately 500 ml. for man.

PRELIMINARY OBSERVATIONS IN DOGS

It was established that 0.05 ml./kg. of Indoklon invariably produced a seizure in dogs upon inhalation. Doses of 0.2 ml./kg. of Indoklon solution injected into the saphenous vein produced seizures in most animals, but generally with latent periods of 30 to 90 seconds. It occurred to us that the long period of latency and failure to produce seizures in certain animals might be due to the hemodilution of the agent prior to its reaching the target cells. Accordingly the injections were made into the external jugular vein. The latent period was reduced to 10 to 20 seconds and there were no failures. Realizing that this procedure was impractical in man, injections were made into the cephalic vein. With this procedure we were able to duplicate the results obtained by the jugular vein injections.

We have established that Indoklon vapor when inhaled is eliminated in the exhaled air. To ascertain the avenue of excretion when injected intravenously, a small cellophane bag containing a mouse was placed over the face of a dog prior to injection of Indoklon solution. At the moment the dog convulsed the mouse likewise showed marked seizures from the exhaled Indoklon. This was repeated on four dogs.

FURTHER STUDIES IN DOGS AND MONKEYS

In three dogs it was demonstrated that the daily intravenous injection of Indoklon solution for 10 days did not produce hemolysis of red cells. Blood studies of these three animals indicated no abnormal variations in hemoglobin or red and white cell counts. The hematocrit, however, did increase following treatment, from a mean control value of 50.2 to a mean post treatment value of 59.4. This indicated some hemoconcentration. The bromsulfalein liver function values were unaffected by the 10-day daily intravenous Indoklon injections. The average latency, *i.e.*, onset of convulsion following Indoklon injection, was 10 seconds, the average duration of the convulsive phase was 68 seconds, while the average recovery period, *i.e.*, time from onset of convulsion to recovery of locomotion, was 140 seconds. The number of convulsions varied between one and two per injection. The behavior of each animal was normal

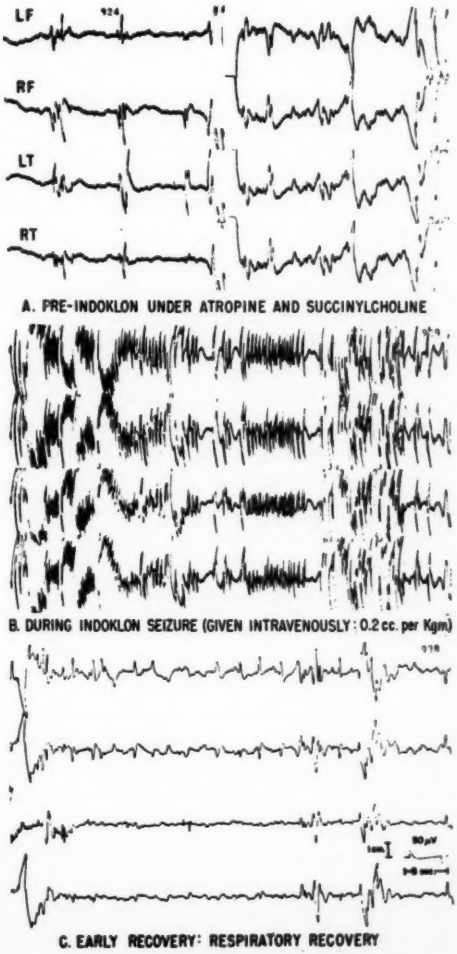


FIG. 1. Cortical dysrhythmia produced by intravenous Indoklon in the monkey.

following each seizure and at the termination of the treatment period. Two dogs survived with impunity five times the convulsive dose of 0.2 ml./kg. The intravenous administration of Indoklon was found to be compatible with premedication with atropine sulfate in the dog and monkey. The procedure evoked the typical cortical dysrhythmia when succinylcholine chloride was used for muscle relaxation. This is shown in the monkey in Figure 1.

PRELIMINARY TRIALS IN MAN

Having completed these pharmacologic studies, one of us (A. A. K.) administered Indoklon solution intravenously to a series of five patients. The dosage was extrapolated from the animal experiments, namely, 0.2 ml./kg. of Indoklon solution. This level and often less never failed to produce the typical seizure in man. The latent period was approximately 20 seconds. The pharmacologic syndrome was similar to that elicited by the inhalation of Indoklon vapor described in detail elsewhere (2). The salient features are twitchings of the orbital muscles, unconsciousness, clonic-tonic seizure, clonic muscular jerks, and recovery within five to 10 minutes. None of the patients could recall the series of events nor did they appear apprehensive about subsequent treatments. There appeared to be no injury to the tissues at the site of injection.

Since polyethylene glycol 300 is dependent upon the kidney for its excretion, we suggest that Indoklon solution containing this agent not be administered to patients with renal insufficiency.

SUMMARY

Solution of Indoklon, five per cent by volume, was shown in laboratory animals and in man to be a satisfactory intravenous method of administering this drug. The results achieved appeared to be comparable in all respects to the inhalation technique originally employed.

REFERENCES

1. Burkhardt, L. Die intravenöse Narkose mit Aether und Chloroform. München med. Wchnschr., **2**: 2365–2369, 1909.
2. Carpenter, C. P. and Shaffer, C. B. A study of the polyethylene glycols as a vehicle for intramuscular and subcutaneous injection. J. Am. Pharm. A., **41**: 27–29, 1952.
3. Esquibel, A., Krantz, J. C., Jr., Truitt, E. B., Jr. and Kurland, A. A. The use of hexafluorodiethyl ether (Indoklon) as an inhalant convulsant. Am. J. Psychiat., **114**: 461, 1957.
4. Kalinowsky, L. B. Personal communication.
5. Krantz, J. C., Jr., Esquibel, A. J., Truitt, E. B., Jr., Ling, A. S. C. and Kurland, A. A. Hexafluorodiethyl ether (Indoklon): an inhalant convulsant—its use in psychiatric treatment. J. A. M. A., **166**: 1555–1562, 1958.
6. Krantz, J. C., Jr., Truitt, E. B., Jr., Ling, A. S. C. and Speers, L. The pharmacologic response to hexafluorodiethyl ether. J. Pharmacol. & Exper. Therap., **121**: 362–368, 1957.
7. Smyth, H. F., Jr., Carpenter, C. P. and Weil, C. S. The toxicity of the polyethylene glycols. J. Am. Pharm. A., **39**: 349–354, 1950.

BRIEF COMMUNICATION

A COMPARATIVE STUDY OF HEXAFLUORODIETHYL ETHER (INDOKLON) AND ELECTROCONVULSIVE THERAPY

ALBERT A. KURLAND, M.D.,[1] THOMAS E. HANLON, PH.D.,[1] AUGUSTO J. ESQUIBEL, M.D.,[1] JOHN C. KRANTZ, JR., PH.D.[2] AND CAROLYN S. SHEETS, B.S.[1]

In a series of studies relating to the anesthetic properties of fluorinated ethers (5) it was found that hexafluorodiethyl ether (Indoklon) consistently produced a convulsive seizure when administered by inhalation to laboratory animals and man. Further experimental investigations (1, 2, 4) indicated that it could safely be used as a convulsant treatment procedure. As a result, it has become of interest to compare a large series of patients receiving Indoklon and electroconvulsive therapy in the same clinical setting to determine whether or not Indoklon has any particular merits over ECT.

METHOD

This study involves various subsamples from two parent groups of 90 patients each, composed of new admissions or readmissions to the admitting service of the Spring Grove State Hospital. One group received a total of 971 Indoklon treatments and the other group received 933 electroconvulsive treatments. The average course for both treatment groups was, therefore, approximately 10.5 treatments, with treatments for the majority of patients spaced at three per week. The selection of the type of treatment was done on an alternate basis. The patients were both males and females between the ages of 18–68 whose diagnoses fell into the following general categories: 1) depressive reactions, both psychotic and neurotic; and 2) schizophrenic reactions for which convulsive therapy was recommended as a symptomatic treatment. All of the patients in the study were referred for convulsive therapy by their ward physicians.

The clinical effectiveness of Indoklon and ECT for two groups of patients from the parent populations (66 patients in each group) were compared on a global psychiatric rating scale immediately after treatment and after a period of three months. A comparison of the incidence of complications was made on all of the patients in both parent populations.

Differential treatment effects were also measured by means of the Multidimensional Scale for Rating Psychiatric Patients, Interview Section (average inter-rater reliability 0.79 for the three raters involved) (6), and, when the patients' conditions permitted, by the Wechsler Memory Scale, Forms I and II (7) and the Graham-Kendall Memory for Designs Test (3). Only those patients who received 12 convulsive treatments spaced at three treatments per week were selected for psychological evaluation. Eighty-nine per cent of the 37 Indoklon patients and 88 per cent of the 33 ECT patients so selected had been in the hospital less than three months, and in no case had any pa-

[1] Department of Medical Research, Spring Grove State Hospital, Baltimore, Maryland. Acknowledgments are made to the following for their helpful support and interest in this project: Dr. Isadore Tuerk, Superintendent, and Dr. Bruno Radauskas, Clinical Director, Spring Grove State Hospital; Drs. William Clark, Eugene C. Waterman, and Blanca Gimenez, Psychiatric Residents, Spring Grove State Hospital; Drs. Leonard Scherlis, Robert T. Singleton, and Luis F. Gonzoles, Department of Cardiology, University of Maryland School of Medicine; Dr. Curtis Marshall, Department of Neurosurgery, Johns Hopkins Hospital; and to the Smith, Kline & French Laboratories, Inc., Philadelphia, Pennsylvania, and the Friends of Psychiatric Research, Inc., Baltimore, Maryland.

[2] Department of Pharmacology, University of Maryland School of Medicine, Baltimore, Maryland.

tient been in the hospital longer than nine months. The mean age was 42.3 for the Indoklon group and 42.0 for the ECT group. At pre-treatment evaluation no significant differences between Indoklon and ECT groups were found to exist for any of the psychological measures, so that the two groups could be considered equivalent in terms of severity of illness and memory functioning. The psychological instruments were administered immediately prior to treatment and on the day after the last treatment by a psychologist who was not informed of the specific treatment involved in each case. Final evaluations were made after a three week follow-up period on those individuals receiving no somato- or chemotherapies subsequent to their 12 treatment series.

Electroencephalographic studies were carried out on a small number of cooperative patients from both parent groups, records being obtained before treatment and at varying intervals after treatment had ceased. Cardiovascular studies comprising serial electrocardiograms and oximetric studies were also obtained just prior to each treatment, during treatment, and immediately afterwards on certain unselective samplings from both groups. The oximetric determinations were obtained by the use of a single scale Waters-Conley Ear Oximeter immediately prior to and during the convulsion.

OBSERVATIONS

In terms of global psychiatric ratings both Indoklon and ECT apparently brought about the same degree of clinical change in the two groups of 66 patients studied. Immediately after treatment 41 of the Indoklon and 36 of the ECT patients had shown moderate to marked improvement. At three months follow-up, 40 of the Indoklon and 45 of the ECT patients were rated in these same categories. At the conclusion of the study 39 Indoklon and 40 ECT patients had been released from the hospital.

The complications observed in both groups of 90 Indoklon and 90 ECT patients seem to be about the same, except for the fact that there were three deaths in the ECT group. Whether the occurrence of the deaths was coincidental with ECT or had some direct relationship to the electroconvulsive therapy, could not be determined. One of the cases expired after her first treatment, and a diagnosis of acute glomerulonephritis was subsequently made. The second patient received only six ECT treatments since she seemed to become more, rather than less, disturbed with treatment. Further diagnostic studies indicated a thyrotoxicosis and three weeks after her last treatment the patient expired. In the third case the patient developed massive diarrhea shortly after his his seventh ECT treatment and died two days later. Jaundice occurred in two cases receiving ECT and was associated with the previous administration of a phenothiazine tranquilizer. The initial enthusiasm that the more gradual onset of the Indoklon convulsion might tend to lower the incidence of fracture was not borne out, since approximately the same number of fractures occurred with both treatment procedures.

The pre- to post-treatment reduction in severity of illness score on the interview section of the Multidimensional Rating Scale was significant at the 0.001 level for both Indoklon and ECT groups; however, an analysis of the covariance of the data revealed that the two treatments were not achieving significantly differential results ($F = 0.02$, 1 and 67 df, $P > 0.10$). Patients from both groups evaluated three weeks after treatment showed further significant reduction in severity of illness scores (0.05 level) but again no difference between treatments was noted.

Changes in Wechsler Memory mean scores for both Indoklon and ECT groups showed approximately the same signifi-

cantly impaired memory functioning ($F = 0.10$, 1 and 56 df, $P > 0.10$). At the end of the three week follow-up period memory functioning was approximately the same as at pre-treatment for patients from both treatment groups, with no significant differences noted between treatments. Results on the Graham-Kendall test were in essential agreement with the Wechsler findings. For those patients studied, neither the post-treatment nor follow-up differences between treatments reached significant levels.

The effect of Indoklon was observed on the EEG's of a group of 10 patients who had normal EEG's before treatment. Records for six of these patients were obtained within a week after treatment and all were found to be abnormal. A third record, which was available on only one of these patients, was found to be normal after a seven week interval. Records for the remaining four patients were available at 11 days, two weeks, two months and five months, respectively, and were all found to be normal at these times. A second group of five patients, whose baselines had been found to be stable in two to three pre-treatment records, was followed by EEG's at weekly intervals until three weeks after the series of Indoklon treatments. Slowing in the theta range occurred as early as the fourth treatment in one patient. The others showed slow activity in the theta and delta range after the fifth, sixth, seventh, and eleventh treatments, respectively. After the termination of treatment, return to the pre-treatment EEG was observed in three patients at two, three, and four weeks. The other two cases still showed slow activity three and five weeks after treatment.

Cardiovascular observations involved a total of 63 patients, 32 of whom had Indoklon and 31 ECT. On the patients receiving Indoklon, 157 tracings were obtained, while 164 tracings were obtained on the ECT patients. A control EKG was obtained immediately prior to the induction of each form of convulsive therapy. Recording was stopped as soon as the shock was given and was resumed as soon as the tremor following the convulsion ameliorated to the extent of permitting the recording of an EKG. Results indicate that the cardiac rate increased in both groups after the convulsion. With an increase in rate there was occasionally seen a slight depression of the ST Segment or lowering of the T wave. Occasional premature ventricular beats occurred in each group. In no instance among either the Indoklon or ECT treatment patients did an arrythmia persist for more than a few minutes. A normal EKG was always recorded 15 minutes after treatment.

The oxygen desaturation results are based on 55 determinations from 10 patients in the Indoklon group and 42 determinations from 24 patients in the ECT group. In only six of the 55 Indoklon determinations were there readings indicating a desaturation of greater than 5 per cent from the beginning baseline. In the ECT group there were 28 determinations indicating a desaturation of greater than 5 per cent from the baseline. This period of desaturation usually began immediately following the clonic phase of the convulsion. It was noted that the average time of the period of desaturation is about 10 seconds with an almost immediate (four seconds) return to the baseline following the convulsion. The significance of these initial differences has as yet not been determined.

CONCLUSION

From the observations reported above it would appear at this time that Indoklon has no definite advantages over ECT. It does appear, however, that Indoklon has the potential for being a safe alternate treatment procedure. Indoklon seems unique in that it offers an almost self-regulating system for determining the amount of convulsant needed by the patients since at the threshold of convulsive action the onset of apnea her-

alds the convulsion, and the administration of the convulsant is stopped. The decreased degree of oxygen desaturation as indicated by the oximetric studies points to the possibility of Indoklon's offering less of a hazard to an already damaged myocardium. What light the use of Indoklon may shed on theoretical considerations relating to the efficacy of convulsive therapy and the mechanism of its action is yet to be determined.

REFERENCES

1. Esquibel, A., Krantz, J. C., Jr., Truitt, E. B., Jr. and Kurland, A. A. The use of hexafluorodiethyl ether (Indoklon) as an inhalant convulsant. Am. J. Psychiat., **114:** 461, 1957.
2. Esquibel, A., Krantz, J. C., Jr., Truitt, E. B., Jr., Ling, A. S. C. and Kurland, A. A. Hexafluorodiethyl ether (Indoklon)—Its use as a convulsant in psychiatric treatment. J. Nerv. & Ment. Dis., **126:** 530–534, 1958.
3. Graham, F. K. and Kendall, B. S. Performance of brain-damaged cases on a memory-for-designs test. J. Abnorm. & Social Psychol., **41:** 303–314, 1946.
4. Krantz, J. C., Jr., Esquibel, A., Truitt, E. B., Jr., Ling, A. S. C. and Kurland, A. A. Hexafluorodiethyl ether (Indoklon) an inhalant convulsant—Its use in psychiatric treatment. J. A. M. A., **166:** 1555–1562, 1958.
5. Krantz, J. C., Jr., Truitt, E. B., Jr., Speers, L. and Ling, A. S. C. New pharmacoconvulsive agent. Science, **126:** 353–354, 1957.
6. Lorr, M. Multidimensional scale for rating psychiatric patients: Hospital form. Veterans Admin. Techn. Bull., TB 10-507, 1953.
7. Wechsler, D. A standardized memory scale for clinical use. J. Psychol., **19:** 87–95, 1945.

HUTT, MAX L. AND GIBBY, ROBERT G. *Patterns of Abnormal Behavior.* Allyn and Bacon, Boston, 1957. xvi + 452 pp., illus. $8.65

In the introduction to their book Hutt and Gibby place considerable emphasis on general educational rather than professional objectives. Their goal is a textbook for the general undergraduate student body rather than for specialized professional students. They therefore make no pretense at completeness in their survey of the field. Instead, they emphasize general principles and concepts of abnormal behavior, illustrating these only with problems that occur relatively frequently or that they consider essential to an understanding of behavior pathology. In general, this approach makes the book parsimonious in organization, clear, readable and teachable.

Hutt and Gibby outline the following overall plan of the book. "Human behavior and deviation in such behavior are present from birth to death. Our plan of discussion is to introduce the reader to some basic concepts, illustrating these in examples of human reactions, then to place these ideas in a historical perspective, to present a theory of the development of human behavior, and finally to treat central examples of deviant behavior as they are observed from infancy through old age" (p. xiii). In accordance with this overall plan the first four chapters deal with basic concepts and history, and the remaining fourteen chapters with the content of the subject.

Among introductory textbooks the present volume is somewhat unusual in its rejection of eclecticism. The authors announce at the outset their allegiance to "modern dynamic theory of personality . . . and the psychoanalytic approach to behavior." To be sure, they do not avoid a brief presentation of other theoretical positions; nor do they hesitate to present clinical and research findings that are inconsistent with their central orientation. On the whole, however, their theoretical chapters constitute a concise introductory presentation of psychoanalytical theory and psychoanalytical ego psychology. Many college teachers will undoubtedly oppose this approach on both theoretical and pedagogical grounds. Certainly such a frank presentation of a single point of view runs counter to tradition in the writing of introductory textbooks. The predominant attitude appears to be that beginning students should be given a representative sampling of various viewpoints, and a thorough survey of the content of a field. Implicit in this traditional approach is the image of the student as a passive consumer of textbook content. To this reviewer it appears that Hutt and Gibby have followed a less traditional but much more vital course. In their very able exposition of the psychoanalytical viewpoint they have provided the student with a consistent frame of reference, imparting to their volume a cohesiveness rare in introductory textbooks. Moreover, the authors emerge as real people with a point of view that they present with conviction, thus avoiding the sterile anonymity one encounters so frequently in prescribed undergraduate reading.

In their discussion of many perplexing problems the authors are able to achieve professional sophistication and didactic simplicity at the same time. One case in point is the consideration of a rationale for the classification of behavior disorders in the chapter on "Deviant Behavior in Children." They write, "There is, however, no natural or inherent system of classification that will serve all purposes equally well. If we agree to base our system on the symptoms or reactions that individuals show, for example, we shall find that people with the same symptoms have different kinds of problems . . . If on the other hand we attempt to devise a

system of classification based upon such concepts as causes, or dynamics or defense mechanisms, our problems are not greatly simplified, if indeed they are not complicated even more. The same cause may produce different symptoms in different individuals with different constitutions, environments, conditions or learning opportunities. Moreover, causation is difficult enough to determine in general, or for a group of symptoms; it is even more difficult to determine in a single instance. Finally, there is usually no single cause for a given reaction; causation is usually complex and there may be interaction within the several component causes" (p. 133).

The authors then go on to propose that behavior disturbances be classified on the basis of ego characteristics and functions, which they spell out in detail in the rest of the chapter.

Although this volume represents an impressive and largely successful effort to present a complex conceptual system and subject matter field to beginning students, it is not entirely free of some common textbook failings. There seems no good way to avoid having textbooks for beginners written by people who have acquired a considerable level of knowledge in a field. Such people are often driven by a completeness compulsion frequently resulting in a characteristic type of over-condensation and shredding of concepts and content. The material tends to be subdivided and re-subdivided under multiple headings as though it were being parcelled out in fragments for rote memory retention. This type of textbook writing is a consequence in part of traditional teaching practices in which large classes are prepared for examination hurdles. It also results from the reluctance of many textbook authors to express their own point of view.

Although Hutt and Gibby cannot be accused of this type of reluctance, they are sometimes led into jargonistic over-condensation by an excessive concern for completeness. The following passage illustrates this tendency. "A distinction is sometimes made between successful and unsuccessful repression. In the former case the individual utilizes *sublimation* (another defense) to discharge the drive effectively, as when the Oedipus situation is resolved successfully and some of the surplus energy of the sex drives is sublimated in other than specific sexual activity. There may also be repression of material (*repression proper* as distinguished from other forms which are known as *primal repression*). In such cases a constant charge of energy is needed to prevent the emergence into consciousness of the repressed material (this is called *counter cathexis*)" (p. 105). In this instance and in some others the authors slip into the textbook writer's malady of saying too much too briefly, and consequently constructing a mosaic of terminology, probably incomprehensible to many of the consumers of the book.

Instances of this kind are fortunately heavily overbalanced by the chapters that develop broad themes in a clear and readable manner. On the whole Hutt and Gibby have successfully attained their objective of writing a readable textbook about a highly complex subject for the student with general educational rather than professional goals.

Benjamin Pope

MEDUNA, L. J., ED. *Carbon Dioxide Therapy*. 2nd ed. Charles C Thomas, Springfield, Ill., 1958. xii + 541 pp., illus. $14.50.

This symposium representing the efforts of the Carbon Dioxide Research Association, sets forth the most recent summary of and developments in data relative to a specialized treatment of nervous disorders—a treatment presumably derived from neurophysiological principles. Without intending it as any part of the criticism of the book, it should be pointed out immediately that any psychoanalysts who read it should do so only after being thoroughly tranquilized,

and would be well advised to conceal it as they pass the doors of the local analytic institute.

The problem of effects of CO_2 on behavior is unquestionably of great interest. That any real understanding and assessment of what it does, if anything, is extremely difficult to obtain is also true. As a therapeutic technique CO_2 administration is beset with the same questions as any other procedure. Similar doubts arise both in connection with diagnoses and assessment of the patient's status and evaluation of any change in the latter, particularly relative to the CO_2 gas itself. Apart from these perennial anxieties, however, we are faced in this instance with a specialized area of problems dealing both with the general topic of somatic therapy and with the physiological factors directly related to effects of carbon dioxide.

The neurophysiological effects of CO_2 appear to depend in a critical way on its concentration in neural tissue, and since elaborate mechanisms involving cerebral vasoconstriction and vasodilatation exist to keep such concentrations relatively constant it is somewhat difficult to make inferences about the mechanisms of action of "low" or "high" concentrations of CO_2 when these terms refer to the composition of the gas mixture inhaled, or to the concentration of CO_2 in peripheral blood. Thus it becomes something of a problem to compare data of different investigators as well as concepts relative to neurophysiological effects. This is not the place for a review of studies in this area, but one wishes that some more impartial integrating analysis had been presented in the symposium under discussion; attempting, at least to clarify the differences in reasoning, observations and concept of several workers such as Gibbs, Gellhorn and Meduna himself. It would seem absolutely necessary to take into account the possible difference in effect of the gas on different cell groups in the brain, (stimulating some and depressing others) and to try to relate this to the observed behavior pattern. Dr. Meduna criticizes Gellhorn for neglecting so-called glandular changes produced by the gas. However he presents no evidence to show that the results of such alterations in glandular secretion can be convincingly related to neurotic states.

Meduna explains neurosis on the basis of pathological reverberating circuits in the cortex. This, in itself, is highly debatable and a good deal more evidence must be obtained to support it as well as possible effects of CO_2 on the neurons involved. The most promising chemical lead appears related to carbonic anhydrase levels if carbon dioxide will cause an increase of this enzyme in cerebral cells. Many points of interest could be discussed here. Suffice it to say that the primary value of the present volume will be to stimulate further laboratory and clinical investigation of this and related problems. Many of the contributions are extremely provocative and it is in this area of stimulation that the value of the symposium lies.

Robert G. Grenell

BOSSELMAN, BEULAH. *Self Destruction.* Charles C Thomas, Springfield, Ill., 1958. xi + 94 pp. $4.75

The primary purposes of a monograph in psychiatry, it seems to this reviewer, are to present new data, to present new ideas, or to present a comprehensive picture of a given field. Does the present volume succeed in any or all of these objectives? Before answering this question it is necessary to take up the question of the definition of self-destruction as used by Professor Bosselman. While the concept is not explicitly defined in the book, the following quotes offer a suggestive definition. "... self-destruction does not usually lead directly to suicide but expresses itself in partial or substitutive ways. Chronic physical illness and disability, neurosis in its manifold forms, drug and alcohol additions, 'martyrdom,' life patterns of repetitious failure, accident proneness, are all, to variable degrees, moti-

vated by this tendency of the human being to turn his aggressive drives upon himself, . . ." (p. vii). "A tabulation of self-destructive activity . . . would be more difficult to compile, including as it would the ego-defeating patterns of sexual delinquency, failures in school and work, and neurotic ill-health, rather than the more overt forms of self-destruction" (p. 34). "Daisy became a prostitute and shoplifter when she was fifteen . . . (She was) lying and stealing at every opportunity . . . But Daisy had begun to destroy herself long before" (pp. 44, 45), (apparently the author is referring to the behavior quoted just above). Since in other places the author suggests that at least certain psychoses are self-destructive, the only conclusion possible is that Professor Bosselman equates self-destruction with most if not all psychiatric disease and symptomatology—neuroses, psychoses, shoplifting, sexual delinquency, lying, prostitution, and failures in school and work—, with accident proneness, and even with physical illness. While in some metaphysical sense the equating of self-destruction and all illness may be true, it hardly seems helpful to the scientific study of death wishes, fantasies about death, attempted suicide, or successful suicide. In fact, this over-generalized use of the term self-destruction seems to the reviewer to be a positive hindrance to careful investigative studies and to practical clinical assessment of individual patients.

To return to the original question, does this book present new data, new ideas or a comprehensive coverage of a field? There are no new data presented in the book. Psychiatry, as of 1959, has progressed far enough that selected case reports, eight of which are described in the book, presented to *illustrate* certain points, cannot be considered data. Second, so far as the reviewer is aware, there are probably no new ideas presented, unless the equating of self-destruction with all or almost all psychiatric illness is considered new. Of course, the question whether new ideas have been presented is partly a question of the reader's perceptions and cannot be answered definitively by any one individual. Third, is the coverage of the literature on self-destruction comprehensive? The author refers to approximately 35 papers and books related to self-destruction, the great majority of which are concerned with suicide or attempted suicide. There is little critical examination of these sources. The author, for example, rarely makes a distinction between successful suicide and attempted suicide in discussing these references or in discussing her own ideas. Since data are available which indicate that there probably are important differences between these two groups, a somewhat unclear picture emerges as to which groups or individuals the author's ideas refer. This is complicated even further by the discussion of patients with death wishes and fantasies of death who do not attempt or commit suicide, since there are almost no data available concerning these kinds of patients.

The basic deficiency in this book is the failure to give psychiatry its due. We have or should have, passed the stage where anecdotes, reflections about individual patients, or incomplete assessment of the state of a given area in psychiatry can be considered significant contributions to psychiatry. It is the belief of the reviewer that this statement applies not only to psychiatry as a science but also to psychiatry as a clinical discipline.

Eli Robins

JONES, MARSHALL R., ED. *Nebraska Symposium on Motivation.* University of Nebraska Press, Lincoln, 1958. x + 278 pp. $3.00

The 1958 *Nebraska Symposium on Motivation* is the sixth in a series of annual volumes dealing with current theory and research in motivation. Each of these volumes comprises six papers which were read during the year at the University of Nebraska.

These symposia provide a rich and varied

fare for the psychologist, psychiatrist, sociologist, and others who share their interests. Physiological psychology, learning theory, personality theory, and psychopathology have been both the initiating stimulus and the incentive for the contributors' efforts. The advanced concepts which the contributors have developed, as well as the practical problems to which they apply them, reveal the depth and breadth of psychology in mid-twentieth century.

Most of the contributors have chosen to present their newest formulations in the context of a review of the pertinent literature. Because of this review feature, the series is a valuable general orientation to the manner in which motivational concepts have been generated from their various sources and applied to specific problems in behavior. One need not be a specialist in behavior theory in order to find this series of greater than ordinary interest; one should, nevertheless, have a professional interest in psychological knowledge.

Throughout this six year period the symposia have provided a distinguished series of contributions. In their attempts to illuminate some particular aspect of the use of motivational concepts in the study of behavior, the participants have either presented new data or resourcefully synthesized the implications of existing data. In this way the series permits the readers to share the newest and most provocative thoughts of some of the ablest and most energetic contributors to the contemporary psychological literature. This feature of the series makes it psychologists' psychology and an important stimulus to conceptual refinements in behavior science.

The 1958 series begins with a refreshing paper by Robert Bolles, who, in a manner characteristic of young men, questions the usefulness of the concept of drive. In his well phrased essay he leads the reader to wonder whether the concept of drive is anything more than a descriptive concept and whether our distinctions between different kinds of drive have any value beyond the descriptive distinctions which they imply. It is apparent that Bolles fears that drives are used to explain the very behavior from which their existence has been inferred. Estes is inclined to believe that the concept of drive can be used with greatest benefit if drives are regarded as the stimuli which initiate and maintain behavior and give it characteristic form. This emphasis of Estes brings into question the traditional concept of drive as an energizing condition or state. There is an attractive simplicity in the supposition that the drive is no more than the stimuli which accompany the states or the circumstances which are ordinarily said to be drive inducing. Spence analyzes the complexity of motivational phenomena and makes us aware of the interaction between the various hypothetical components of a motivated state. In a manner which could have been anticipated from his well known writings, his formulations emphasize the concept of incentive, and among the present contributors he is most distinctive in this regard.

Littman's paper is reminiscent of Bolles' in that it leads us to wonder about the value of a concept of motivation. In his comparative review of the literature he makes the reader aware of the degree to which psychologists have used the concept of motivation as a way of thinking about behavior rather than as something that is thought about behavior. It would seem that our various concepts of motivation may have very little in common other than the fact that they are used by us as aids in the study of behavior. Thus it would appear that in Littman's view we would be little poorer if the word "motivation" were suddenly removed from our vocabulary altogether.

Charles Eriksen's paper is different from the others in that he seems only indirectly concerned with the meaning and value of motivational concepts and is primarily concerned with some of the properties of unconscious processes and the manner in which

awareness may be related to perceptual defenses and other reactions of clinical significance. In the final paper of the 1958 symposium Malmo is concerned with the problem of estimating drive strength and is interested in the possibility of using general physiological arousal as an evidence for drive strength. Malmo, like other contributors to the present symposium, recognizes that the relationship between drive strength and response strength is not a simple one.

Many readers will be simultaneously stimulated and discouraged by the papers of the 1958 symposium. Most of the accepted generalizations about the nature and operation of drives and motives are questioned by these writers, and the resultant feeling of "where do we stand now?" is somewhat discouraging. Beyond this, however, the present series has strongly encouraging implications because it shows the nature and the vigor of the current quest for new understanding. The inadequacies and the inconsistencies of commonly held beliefs are dissected with unusual care, and this scrutiny is accompanied by evidences of resourceful and creative constructive efforts.

It is fully apparent that behavior theory is in the process of change, but the exact nature and direction of this change are not obvious. Perhaps the most significant trend apparent in these symposia is that our present scholars are not inclined to oversimplify or underestimate the complexity of the phenomena which they study. Whether this is the route to better theory remains to be seen.

J. R. Wittenborn